Minnesota Medicine

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

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Minnesota Medicine

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

Volume 34 October, 1951 Number 10

HYPERTROPHIC PYLORIC STENOSIS

HAROLD F. FLANAGAN, M.D.
Saint Paul, Minnesota

MOST of the literature on the surgical treatment of congenital hypertrophic pyloric stenosis gives one the impression that the treatment of this disease is simple and routine. This, however, is not true, for it is a major surgical operation in a partially starved, and metabolically disturbed infant. The baby, at the age surgery is usually done in this condition, is a most labile creature. He is very susceptible to infection, has a most unstable fluid balance, and requires a maximum of nursing care. Therefore, it seemed only natural to choose this subject in order to evaluate the care a pediatric hospital, such as Children's Hospital, Saint Paul, offers to its patients.

The proper care of these patients involves not only the surgical staff but the pediatric and resident staff, the laboratory and roentgenological service, and yet, all of these would be helpless without the nursing staff.

The present study covers the period from 1945 to 1950, inclusive, and includes all of the cases surgically treated for congenital hypertrophic pyloric stenosis. There were seventy-three infants surgically treated. Seventy-two of these had pyloromyotomies. These cases were medically managed by nineteen pediatricians or general practitioners, and the surgery was done by eleven different surgeons. Nine of these surgeons limit their work to surgery; two do general practice but are qualified by the hospital staff to do this type of surgery.

The Children's Hospital is a private hospital, and most of the patients are private patients. The hospital has a "free bed" service for part, or wholly, charity cases. The patients admitted to this service are cared for by the medical and surgical staff on a rotating basis.

The resident staff consists of one affiliating intern, one first-year and one second-year fellow in pediatrics at the University of Minnesota. The laboratory staff is under the direction of Dr. Kano Ikeda, and the roentgenologic service is directed by Dr. Harold Peterson.

The nursing staff is most complete, being well trained and very co-operative.

Of the seventy-three cases treated surgically, seventy-two had pyloromyotomies. At operation, one infant was found to have no pyloric tumor and a patent pyloric canal. After a surgical exploratory, the abdomen was closed, and the infant was treated medically for pylorospasm. Another infant required a second pyloromytomy before the obstruction was relieved. Eight of the babies surgically treated had surgical complications, and nine of them had medical complications, one of them dying from pneumonia. With seventeen of seventy-two patients showing either medical or surgical complications, this treatment is not to be considered lightly. Therefore, while it may be repetitious, it is appropriate to review in some detail the pathologic condition, the signs and symptoms, the preoperative and postoperative care, as well as the diagnosis and complications of congenital hypertrophic pyloric stenosis.

A typical case of congenital pyloric stenosis is described by Szilagyi and McGraw.[14] The infant is usually a lusty specimen, probably the first born in the family, born spontaneously after a normal pre-natal development. He nurses well and shows

Inaugural thesis presented before the Minnesota Academy of Medicine, March 14, 1951.

good progress until the second or third week of life, when, rather suddenly, he begins to vomit some of his feedings. While maintaining his appetite, and without developing a febrile disease, the vomiting becomes gradually more severe, and the infant begins to lose weight, becomes wizened, and, unless energetic medical attention intervenes, he dies of starvation or pneumonia before the age of three months.

Congenital hypertrophic pyloric stenosis is the most common surgical condition in the first three months of life. While the surgical treatment of this disease is good, we cannot let up on the vigilance of an alert medical and surgical team if we are going to advance in the therapeutic management of this disease. Every practicing pediatrician and almost every general practitioner and general surgeon will be called on sometime to treat an infant with hypertrophic pyloric stenosis. It is then that the excellent mortality figures of Ladd,[6] Donovan,[3] Wyatt,[15] and others like them, mean little unless we are prepared to follow their advice completely and meticulously. Ladd states that the mortality rate in a Children's Hospital should not be above 1 or 2 per cent, but to attain such results requires attention to the details of preoperative and postoperative care as well as the operative technique.

Historical

According to Kellet (quoted by Szilagyi and McGraw), the disease was first reported by a German pediatrician, Hildanus, about 1646. Blair in 1717, Weber in 1758, and Armstrong in 1771, reported cases strikingly similar to the disease as now recognized. In 1778, in the first medical periodical published in the United States, Hezekiah Beardsley[12] reported what, for years, was thought to be the first case on record of this disease. It was not, however, until 100 years later, when Hirschspring at the Widsbaden meeting of the "Geselschaff fur Kinderheilkinde" in 1887, reported two cases of infantile pyloric stenosis, that the disease was fully recognized. Hirschspring was primarily interested in the pathological anatomy of this disease and gave an accurate account of the gross anatomy. He supposed that this hypertrophied pylorus covered a typical type of infantile vomiting.

Within a very short time, the disease became widely known. Finkelstein, Huebner, Pfaundler in Germany; Hutchinson and Thomson in England; Holt, Downe and Scudder in the United States were especially eminent among those who early interested themselves in this disease.

Predisposing Causes

A great many factors have been reported to bear a relationship to the incidence of congenital hypertrophic pyloric stenosis. The disease occurs predominately in males. In Ladd's series, 85 per cent of the cases treated were males. This is about the relationship shown by most observers. In our series, 87 per cent were male children.

The disease is reported to occur most frequently in the first-born infant. The usual percentage found is between 50 and 60 per cent. In the cases reviewed in this group, 39 per cent occurred in the first-born child.

All of the infants in this group were of the white race. Donovan[4] reports 2 per cent of his cases occurred in the negro race, and Romanos[13] reports that 30 per cent of his cases at New Orleans Charity Hospital occurred in the colored race.

Familial occurrence is noted by many observers, among them Sauer, Finkelstein, Ladd, and Bilderback. The twinning incidence of pyloric stenosis is strong in support of a hereditary factor. Laubscher and Smith[8] find pyloric stenosis present in both twins in eleven of thirteen monozygotic twins, but in only two of twenty-four sets of biovular twins. In the seventy-two cases reviewed, there was no history of any other sibling having had surgical treatment for this disease. There were four cases where other children in the family had been treated for persistent vomiting in infancy, and, in one instance, the father of one of the infants had been treated for pylorospasm as an infant.

There was no definite seasonal incidence noted in this group of cases.

Pathology

The primary pathologic lesion is limited to the pylorus. Other pathologic changes, such as wasting of the body, and metabolic disturbances, with alkalosis and death, are all secondary to, and result from, the obstruction caused by the stenosis of the pylorus.

Grossly, there is a smooth shining mass, usually cartilagenous in consistency, at the pyloric an-

tral region. This mass, loosely spoken of as a tumor, is spindle shaped, its length varying from 2 to 3.5 cm. and its width from 1.5 to 2 cm., with a wall thickness of .5 to .8 cm. The muscular coat of the entire stomach is thickened to some degree, and the lumen dilated. The mass itself is formed by a hypertrophy and some hyperplasia of the circular muscle fibers. There may be some involvement of the longitudinal muscle as well. This increased muscular volume within the confinement of an adherent and unstretchable peritoneal covering, transforms the pliable pyloric canal into a hard and solid tube. The swelling of the muscle causes a protrusion of the mass into the duodenum, much as the cervix protrudes into the vagina.

The hypertrophy of the muscle mass causes a compression of the vascular branches, and gives rise to the pale, bloodless appearance of the muscle fibers and component tissue. The consistency of the muscle turns crisp and gritty as the disease progresses and, probably on account of the stretching and loosening of the peritoneal covering by the growth of the muscle fibers, the mass again becomes softened and more vascular. It is this factor of age that apparently accounts for the two types of tumors found by the surgeons.

The tumor mass reaches the maximum hardness and size from the fourth to the ninth week, postnatally. The secondary softening of the muscle mass may account for the tendency of spontaneous recovery seen in some cases.

Microscopically, there is a marked hypertrophy of the circular smooth muscle layer, together with an actual increase in the number of smooth muscle fibers. Sections of the mucosa reveal edema and leukocytic infiltrations.

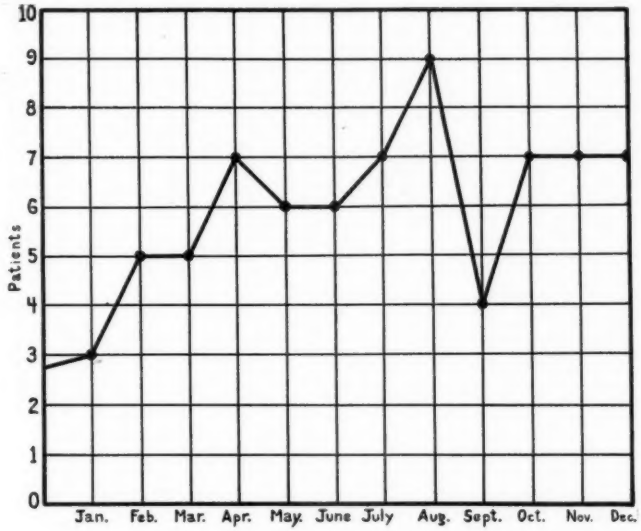

Fig. 1. Seasonal incidence of pyloric stenosis cases studied.

Causes—Theoretical

Many theories as to the cause of this condition are advanced but no one has found a single theory that is acceptable.

That the tumor is present at birth is generally agreed, It has been found in premature and stillbirths. Whether the tumor is a congenital anamoly is questionable. The infreqency of associated congenital anamolies is noted by most of the observers. Lehman offers the theory that the pyloric sphincter, differing from the rest of the musculature of the pyloric canal, is in a state of tone when not working; its working state occurs when the sphincter opens. Thus, hypertrophic pyloric stenosis is a congenital hereditary inability of the pyloric sphincter to open. This is similar to an adhesion of the cardiac sphincter. The result of this condition is a marked hypertrophy of the pyloric musculature.

Ladd believes the disease is a combination of

pylorospasm and a marked hypertrophy of the pyloric muscle, together with edema of the mucous membrane from mechanical irritation of the milk curds. The onset of symptoms, usually between the second and fifth week, the severity of the vomiting, and the ultimate development of complete pyloric obstruction are directly related to the degree of hypertrophy present in the individual case.

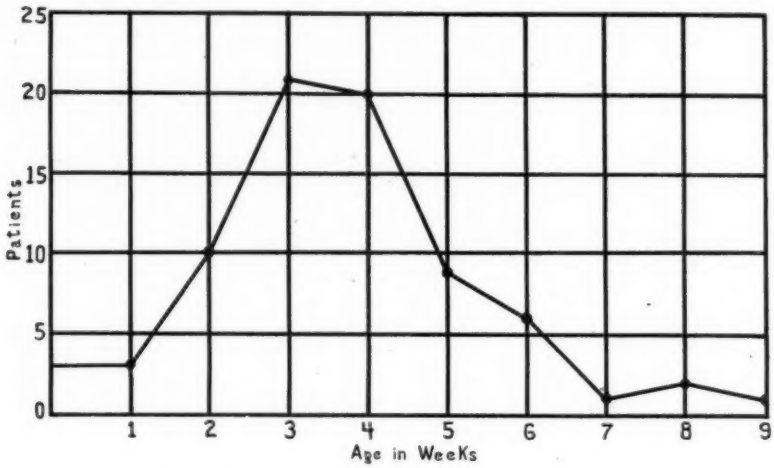

Fig. 2. Comparison of ages at onset of symptoms.

Donovan believes the tumor is congenital; that the pylorospasm follows the hypertrophy, and that the spasm is responsible for the onset of symptoms at two to five weeks of age. He bases his conclusions on the disappearance of the tumor about seven weeks after a Fredel-Ramstadt pyloromyotomy, and the persistence of a tumor mass for twenty-nine years after a posterior gastroenterostomy done for this condition. He believes the congenital origin of the tumor is supported by the predominance in males, the tendency to occur in the first born, as well as the finding of the tumor in premature infants, who failed to survive because of prematurity.

Signs and Symptoms

Vomiting is the presenting symptom in every case. The vomiting usually begins in the first two to three weeks of life. The earliest in the groups was one week and the latest nine weeks. At first, the vomiting is infrequent and not forceful. Usually, in a few days, the vomiting becomes more frequent and projectile in character. The child is continually hungry, and will feed eagerly right after vomiting. The vomitus never contains bile, but it is curdled. Blood in the vomitus is not infrequent. It was grossly present in nineteen of the seventy-two infants.

As the vomiting increases in freqency, there is loss of weight. At the time of surgery, most all of the infants are at, or under, birth weight.

Constipation is a frequent complaint mentioned by the parents. The child is vomiting and having infrequent stools. At first, the stools are small and hard; later they become scanty, mucoid and dark green in color. These are the "starvation type" of stools. Diarrhea seldom occurs in this disease. If it does occur, it is a very serious preoperative complication.

The presence of visible peristaltic waves is clinically characteristic of this disease. With the infant between the examiner and the window, or light, inspection of the abdomen will reveal waves of peristalsis, progressing from left to right in the epigastrium. These waves are large, and as the wave reaches the pyloric region, a characteristic frown may frequently be seen on the face of the infant. Peristaltic waves were noted clinically in all of the seventy-three cases.

Palpation of the abdomen for a tumor mass should be carefully done. The finding of a palpable tumor is almost pathognomonic of this condition. Ladd and Donovan believe a tumor can be felt in almost every case if carefully palpated for. Grimes, Bell and Olney[5] find the tumor palpable in about 70 per cent of their cases. In this group, the mass was definitely palpated in thirty-

one infants and questionably palpated in sixteen; it was not felt in twenty-six. At surgery, there was a definite tumor mass found in seventy-two of seventy-three cases.

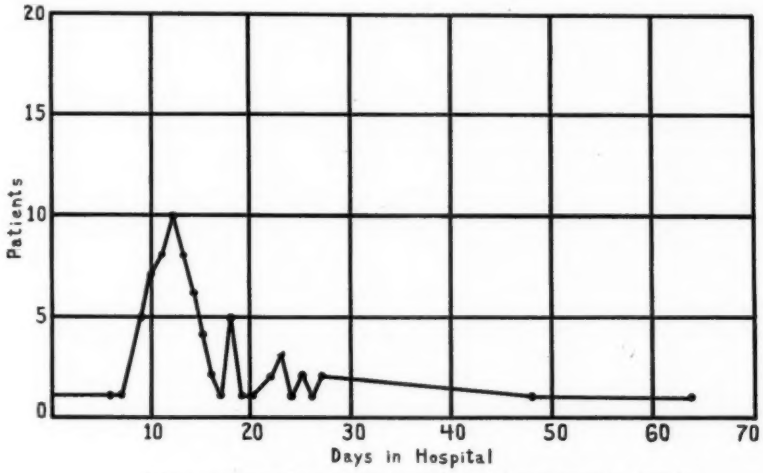

Fig. 3. Comparison of total number of days spent in hospital.

Dehydration is the most serious symptom of this disease. It is important to recognize this dehydration and absolutely necessary to correct it before operation if we are going to avoid losing unnecessary lives. If the infant is followed closely and the diagnosis made early, this symptom can, in a large measure, be prevented. It is most important to recognize the moderate from the severe type of dehydration.

The child suffering from moderate dehydration has, and is making a quite satisfactory adjustment to the loss of fluids. The vital functions, especially of the brain, kidney and liver, have not, as yet, been impaired. On the other hand, the severely dehydrated infant has exceeded his physiologic compensatory mechanism, and organic damage to the brain, kidney, liver and spleen may, or does occur. This damage is the result of lack of oxygen, due to a reduction of the blood flow and hemo-concentration.

The child with moderate dehydration is anxious and irritable; the skin is dry; the mucous membrane of the mouth is still moist. The skin of the axilla, groin, hands and feet is still moderately moist. The blood protein and urea nitrogen values are normal. The urine is scanty and has a high specific gravity. There may be some fever, particularly in the warm weather.

The infant with severe dehydration has a grey, or ashen color; the skin is dry, leathery, and occasionally mottled. The skin may be so dry it lies in folds about the buttocks. The eyeballs are soft and sunken. Instead of being irritable, the child is apathetic. The mucous membrane of the mouth is dry and the saliva thickened. Anuria is usually present. The hemoglobin and plasma proteins are elevated, indicating hemo-concentration. The urea nitrogen of the blood is elevated.

Dehydration in this disease is the result of vomiting, due to pyloric obstruction. As a result of vomiting, there is a loss of hydrochloric acid; this leads to a depreciation of the blood chlorides, and a compensatory increase of the bicarbonate of the blood. The wastage of ingested food results in a depletion of the protein and water. Thus, besides dehydration in this disease, there may be a protein deficiency and an alkalosis.

Alkalosis manifests itself in the form of irregular, shallow breathing, with frequent periods of apnea. Tetany may occur. Tetany in most of the cases manifests itself by muscular rigidity and occasionally by convulsions. The presence of alkalosis can be confirmed by an analysis of the carbon dioxide content of the blood serum for the carbon dioxide content of the blood serum is largely a measure of the blood bicarbonate.

Akin, Jr., and Forbes,[1] in a clinical and laboratory study, show a definite correlation exists between the duration of vomiting and an increase in the carbon dioxide content of the blood serum. The length of time vomiting continues is likewise correlated with the chloride loss in the blood se-

rum. In the same series of cases, they also show the effects of vomiting and dehydration on kidney function. In thirty-six cases, twenty had elevated non-protein nitrogen values; ten of these were in the range of 35 to 50 mg. per cent; nine between 50 to 100 mg. per cent and one 112 mg. per cent.

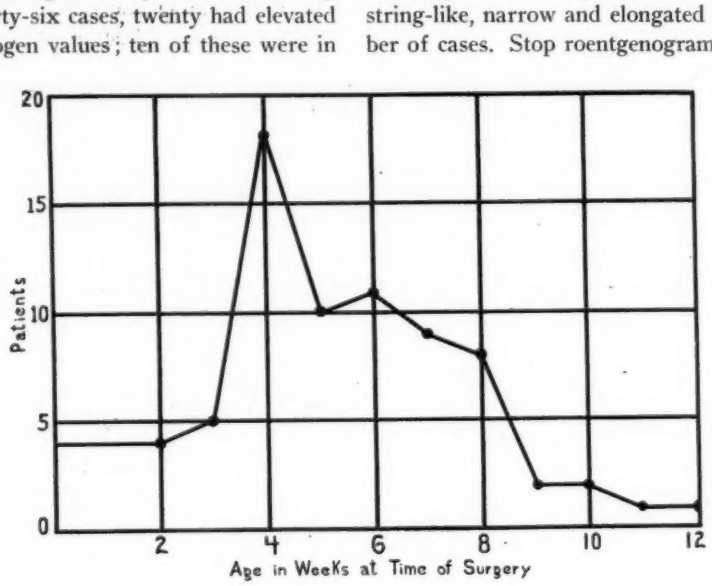

Fig. 4. Comparison of ages at time of surgery.

Roentgenologic Findings

In most cases, roentgenologic examination is not necessary in arriving at a diagnosis, but in the atypical or questionable cases, it is most valuable. There is no contraindication to a proper roentgenologic examination. In this group of cases, the roentgen examination was the diagnostic media in one infant, and, had it been used in another, it would, very likely, have prevented unnecessary surgery.

At roentgen examination, the stomach should be completely emptied, by aspiration, to remove air and gastric fluid. Then, a thin barium mixture of 1 to 2 ounces should be introduced into the stomach by tube. The infant should be examined fluoroscopically in the lateral and oblique, as well as the anterior and posterior, positions. The oblique view is particularly valuable in bringing the pyloric canal into the field.

For some time, the roentgenologic criteria have been the dilatation of the stomach and a delay in the emptying time. These are valuable, but not diagnostic findings.

Olnick and Weens,[11] using a technique where they gently press the contrast media into the pyloric region, find it possible to demonstrate the string-like, narrow and elongated canal in a number of cases. Stop roentgenograms in the oblique and prone position often demonstrate this finding clearly. The unfailing roentgen evidence of this disease is the demonstration of a long, narrow, string-like shadow, only 1 to 2 mm. in width—the so-called "string sign." The pyloric beak is another rather definite diagnostic x-ray finding. Occasionally, indentation of the duodenal cap can be demonstrated; this, too, is diagnostic. These may be called the specific signs. It is the impression of the reviewer that the roentgenologic examination was not used sufficiently in this group of cases.

Differential Diagnosis

In most cases, the disease is so clear cut that the differential diagnosis is not difficult. Pylorospasm must always be considered in arriving at a diagnosis in these infants. Whether we wish to consider pylorospasm a true disease entity or not, a good number of infants are seen by the pediatrician who, on the first examination, cannot be differentiated from those with congenital hypertrophic pyloric stenosis. Only observation and time, or the response to atropine or the barbiturates, can make the diagnosis definite. For, in pylorospasm, the vomiting is usually controlled by concentrated feedings, atropine, or phenobarbital,

within four or five days. In one of the cases in this group, in spite of hospital observation, diet and sedatives for five days, a diagnosis of hypertrophic pyloric stenosis was made and operation advised. On opening the abdomen, there was no tumor found and the pylorus was patent. This was a case of pylorospasm. Nafe[10] reports similar experiences.

Vomiting that is due to infection can usually be differentiated by a careful physical examination. It seldom affords any diagnostic problem.

Intracranial injuries may cause vomiting that simulates that found in hypertrophic stenosis of the pylorus. A good history and thorough physical examination is almost always sufficient to warrant a differential diagnosis.

Esophageal atresia and stenosis, and occassionally esophagitis, may produce vomiting and loss of weight, two of the common symptoms of congenital hypertrophic pyloric stenosis. However, in atresia or stenosis of the esophagus, vomiting is usually present since birth, the vomitus is not curdled and vomiting usually occurs at, or almost right after, feeding. The inability to pass a catheter into the stomach is the easiest and surest differential in these conditions. In the infants with esophagitis, vomiting may not begin for from two to five weeks after birth, but the type of vomiting is much the same as that found in atresia or stenosis of the esophagus. Roentgenologic examination is most helpful in making this diagnosis.

Congenital obstruction of the first part of the duodenum may produce symptoms similar to those seen in hypertrophic pyloric stenosis. However, in duodenal obstruction, the vomiting is present since birth and the vomitus is bile stained. X-ray examination will usually make this condition apparent.

Extrinsic intestinal obstruction, such as malrotation of the colon, or congenital bands that cause pressure in the duodenum, may give rise to explosive vomiting, and visible gastric waves. The vomitus usually contains bile and the roentgenologic examination is diagnostic in these conditions.

Chalasia of the esophagus is a new syndrome first described by Edward Neuhauser.[2] It may produce vomiting, weight loss and simulate in some degree the picture of congenital hypertrophic pyloric stenosis. In one of the cases in this group, it was present along with hypertrophic pyloric stenosis. In chalasia of the esophagus, the infants vomit persistently, usually beginning about the second week of life. They do not have gastric waves and the vomiting is usually not projectile. Roentgenologic examination reveals a dilated cardia and frequent regurgitation of the barium into the esophagus. The infants with this condition respond to thickened feedings, and although Neubauer suggests feeding and retaining the infant in Fowler's position, it has been our experience they do best when they are fed and kept on their abdomens.

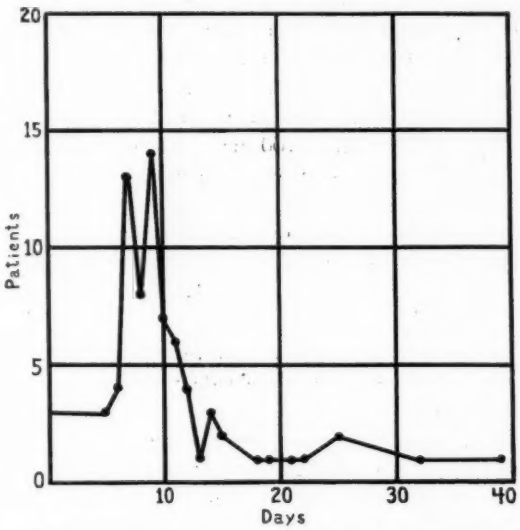

Fig. 5. Number of days spent in hospital postoperatively.

Treatment

Medical treatment of congenital hypertrophic pyloric stenosis should not be continued beyond the time necessary to differentiate the probable from the established cases. If this policy is established, the dangers of dehydration are eliminated and the prognosis improved.

When the diagnosis has been made, the infant should be hospitalized for preoperative therapy. If the infant has no infection, he should be admitted to a clean nursery, and isolation nursery technique adhered to. If he has any evidence of a complicating infection, he should be isolated and the infection vigorously treated with antibiotics. At the same time, his fluids should be maintained. It is well in all of these cases to

have a chest x-ray at the time of admission to the hospital.

Preoperative treatment should be aimed at overcoming the fluid loss, a replacement of the liver glycogen reserve, and the establishment of a normal electrolytic balance. When, or if the kidney is functioning normally, the protein deficit should be replaced with plasma or whole blood. Vitamin C should be given in the intravenous fluids in amounts of 100 mgm. per day, and vitamin K should be given preoperatively for two days. The ideal preoperative treatment is two to three days. This condition is never a surgical emergency. More infants are lost from inadequate preoperative therapy than from surgical errors.

The amount of time required to prepare the patient for surgery is dependent on the state of dehydration. Ladd gives two intravenous infusions and two subcutaneous clysis daily. The intravenous solution he uses is 5 per cent dextrose in distilled water, given in amounts of 10 cc. per pound of body weight. The subcutaneous solution is either isotonic, or one-half isotonic saline in 2.5 per cent dextrose, and in amounts of 15 cc. per pound of body weight.

The infants in this series were treated with a variety of fluids: saline, Ringer's lactate, 5 and 10 per cent glucose, one-third Darrow's solution and Butler's solution. During the first two years of the period reviewed, the fluids were given in a majority of cases by subcutaneous clysis. The past four years, intravenous administration of preoperative fluids has become almost routine. There was only a small amount of the total fluids given by the subcutaneous route in the past four years.

The most satisfactory hydration and replacement fluid used in these cases is one prepared from a mixture of Butler's solution and 5 or 10 per cent glucose in distilled water.

Lowe et al[9] have shown the value of potassium therapy in the surgical patient and its role in preventing chloride loss. Their studies show that in the patient suffering fasting, dehydration and intracellular potassium depletion due to an alarm reaction, or to an alkalosis resulting from the loss of gastric chlorides, the administration of isotonic sodium chloride may be ineffective in accomplishing extracellular repair due to sodium going with the water to the dehydrated and potassium depleted cells, and chloride being excreted in the urine. When such therapy is modified by decreasing the ratio of sodium to water, and adding potassium, there is a more physiologic hydration of both intra- and extracellular fluid compartments of the body. The chloride is conserved by the kidney, and the sodium tends to stay in its proper extracellular position.

Butler's solution[7] is prepared as follows:

66 grams or 589 cc. of molar sodium lactate solution (100 cc. or 40 cc. ampules may be used)
30 grams of KCl
18 grams of NaCl
15 grams of K_2PO_4
1500 cc. freshly distilled water
Filter through a glass filter; bottle in 50 cc. bottles and autoclave

When the patient is admitted to Children's Hospital, his fluid requirements are calculated per square meter of surface area, per twenty-four hour period. The surface area in square meters is obtained by the following formulas:

$$\text{Surface area in sq. meter} = \sqrt[3]{(\text{wt. in kg.})^2} \times 0.1$$

The infant's basic fluid requirement is taken as 2400 cc. per square meter per 24 hours; 3000 cc. per square meter per 24 hours is the replacement fluid required in the first day of preoperative preparation. Roughly, this is about 150 cc. per kilogram of body weight per 24 hours.

A total of 360 cc. per square meter of a so-called hydrating solution, containing 500 cc. normal saline and 1000 cc. of 10 per cent dextrose in distilled water, is given intravenously in the first forty-five minutes or until the infant voids. If the infant does not void in forty-five minutes, the rate is slowed to ten to twenty drops per minute until he does void. Then a fortified polyionic solution made with:

50 cc. Butler's solution
200 cc. normal saline
800 cc. of 10 per cent dextrose in distilled water

is substituted for the hydrating fluid and given by continuous venous drip, in the amount previously determined, as required in the first twenty-four hours. The next day, unless there is a gastric tube in place, a regular polyionic solution of:

50 cc. Butler's solution
1000 cc. 10 per cent dextrose in distilled water

is given intravenously in amounts of 24000 cc. per square meter per 24 hours.

After forty-eight to seventy-two hours of this therapy, the infant is usually well hydrated and in good preoperative condition. If there is any question of the infant's condition, it can be carefully checked with a determination of the carbon dioxide content of the serum, a serum chloride determination and an evaluation of the urea nitrogen of the blood.

If there has been a prolonged period of starvation and especially if, after hydration, there is a lowered serum protein, the use of plasma or whole blood is most valuable in overcoming the protein deficit. If anemia is present after hydration, whole citrated blood, not in excess of 10 cc. per pound of body weight, should be given preoperatively.

The infant is fed until six to eight hours before surgery. There is only a rare case in which some food does not get through the stenosed pylorus into the intestinal tract. The stomach should be emptied one-half hour before operation; then a No. 10 ureteral catheter is passed through the nose and into the stomach and taped in place. This catheter is left in the stomach during the operation. It is a convenient aid in deflating the stomach, and it may be useful in this manner to both the anesthetist and the surgeon.

Preoperative medication, excepting atropine, is not warratned in these cases. Atropine is given one-half hour before operation, and the dosage is determined according to the age of the infant.

It is routine in the hospital to wrap the infant's extremities in sheet wadding before taking him from his room to the operating room. The operating room is kept warm and preoperative preparation of the surgical field is carried out with a minimum of exposure to the infant.

Drop ether is the anesthetic of choice in most cases. Local anesthesia, with novocaine or procaine may be useful in some cases, especially in those with upper respiratory infection. The danger of poor relaxation, sudden straining, and evisceration with resultant shock must be considered in the use of this type of anesthesia. In this group of cases, fifty-one of the pyloromyotomies were done under ether anesthesia, and twenty-one with local anesthesia.

Surgery

The abdomen was opened through a right rectus incision in all of the cases in this series. The Fredel-Ramstadt type of pyloromyotomy was done in each case. Aside from accidental perforation of the duodenum in five of these patients, there was no unusual surgical difficulty encountered. There was no anesthetic difficulty of note in the operated cases. Let me here pay tribute to the anesthetists and the surgeons.

Postoperative Treatment

Postoperative feeding is started as soon as the infant is fully conscious, except in the cases where there has been a perforation of the bowel. In these cases, feeding by mouth is withheld for twenty-four to forty-eight hours, and the infant maintained on a fluid regime similar to that given preoperatively. Many different feeding schedules were used in this group of patients and, in attempting to evaluate them, the nurses' notes were carefully studied and many of the nurses interrogated as to:

1. What feeding the infant liked best.
2. The feeding on which he seemed to have less distress after ingestion.
3. The feedings that seemed to be best retained.

It is the opinion of the reviewer that the whey feeding used in a schedule similar to the one advocated by Ladd[7] is the most satisfactory of those tried.

Whey is prepared by adding five tablespoonsful of liquid rennet, or two or three junket tablets, dissolved in a small amount of water, to a pint of warm milk, heated to 100 degrees F. After the milk has coagulated, it should be placed in a cool place for one-half hour. The curd is broken with a fork, and the whey (liquid) strained through a cheese cloth. The whey is then brought to a boiling point to destroy any remaining enzyme.

The feeding schedule is as follows:

One ounce of fluid is given every two hours after the infant is fully awake.

Two feedings of sterile water.

Four feedings of whey.

Four feedings of one-half whey and one-half breast milk or formula.

Four feedings in which the whey is decreased one dram and the breast milk or formula increased by a like amount.

At the end of twenty-six hours, the infant is getting one ounce of whole breast milk, or formula. In the next four feedings, the breast milk

TABLE I. COMPLICATIONS OCCURRING IN THE CASES OF PYLORIC CASES OBSERVED IN THIS STUDY

Medical		Surgical	
Pneumonia	3	Duodenal Perforations	5
Diarrhea	3	Hemorrhage	2
Upper respiratory with Otitis media	2	Reoperation	1

One pneumonia resulted in death.

or formula is increased by one dram a feeding so that at the end of this regime, the infant is getting 1.5 ounces of breast milk or formula every two hours. The eighteen feedings will require thirty-six hours. When breast milk is not available, Olac, used in normal dilution, was found to be an excellent formula. The infant is fed by bottle and is held while fed; he is burped frequently.

Regurgitation in the postoperative period is not uncommon in the first twenty-four to forty-eight hours. Thirteen of the infants in this group stopped vomiting or regurgitating after the first twenty-four hours following operation. Thirty-one of the infants had no further postoperative difficulty after forty-eight hours. Ten had some regurgitation for three days. Two continued to regurgitate an occasional feeding for four days. One continued with occasional vomiting for five days and two had some regurgitation for six days; another for nine days. One infant did not do well after operation, vomiting some, or all of his feedings for the whole of his postoperative period. He expired twenty-two days after surgery. An infant with chlasia of the esophagus continued to vomit for weeks postoperatively, and was only controlled when the condition was recognized and proper postural treatment instituted.

Complications

The complications in this group of infants were relatively frequent. In five of the cases, the duodenum was perforated at the time of operation. The perforation was surgically closed in each instance. In these cases, antibiotic therapy was instituted at once, fluids by mouth were withheld for twenty-four to thirty-six hours, and in two of the infants, nasal suction was used the first twenty-four hours. Intravenous fluids were given to maintain the infant in fluid and electrolytic balance. In one case where the duodenum had been perforated, the abdominal wound partially broke down one week postoperatively. There was no evisceration, but the wound was resutured and the infant made an uneventful recovery. In another case in which the duodenum was perforated, the abdominal wound became infected on the third postoperative day, about the same time the infant developed a diarrhea, and had a stormy convalescence. The excellence of the fluid therapy, and antibiotics, were responsible for this infant's recovery.

Postoperative pneumonia occurred in three infants; two recovered rapidly with the aid of antibiotics and supportive fluid therapy.

One infant, who vomited persistently postoperatively, died of an aspiration type of pneumonia in spite of fair fluid, and good antibiotic therapy. This infant was a male, born October 4, 1945, and admitted to the hospital on October 30 with a history of vomiting for two weeks. The birth weight was 7 pounds 6 ounces, and the admission weight was 6 pounds 10 ounces. The preoperative fluid therapy was fair. A pyloromyotomy was done under ether anesthesia on November 2. The infant continued to regurgitate two or three times a day, but with the help of supportive fluid therapy, he was doing fairly well until November 16. His weight on that day was 7 pounds 8.5 ounces. On November 17 he became feverish, vomited all of his feedings and, in spite of antibiotic therapy, expired with an aspiration type of pneumonia. This was confirmed by postmortem examination. The infant's fluid therapy from November 17 to November 24 was not particularly good.

Upper respiratory infection with otitis media occurred postoperatively in two infants. Their response to antibiotics was excellent and the recovery was uneventful.

Diarrhea was a postoperative complication in three infants. In one infant, the disease was mild and responded readily to limitation of the diet and antibiotics. Diarrhea in the second infant developed on the tenth postoperative day. It was severe and the child required supportive fluid therapy for one week. He was discharged as cured on the twenty-third postoperative day. The third case of diarrhea occurred in one of the infants who had surgical perforation of the duodenum. This infant may have had a peritonitis. He was very ill for two weeks and needed fluids and antibiotics for

this entire period. He was in the hospital thirty-six days after surgery but was discharged cured, and has remained well since.

Postoperative hemorrhage occurred in two cases. In the first of these, the infant vomited old blood and had black, tarry stools on the second postoperative day. The hemoglobin dropped 2 grams in this forty-eight hour period. Feedings were stopped and he was given a transfusion of whole citrated blood, and supported by intravenous fluids for two days. After this period, he made an uneventful recovery and was discharged on the eleventh postoperative day. In the second case, there was bleeding from a vessel in the abdominal wound, noted an hour after operation. The abdominal wound was reopened and the bleeding vessel tied off.

Reoperation was necessary in one infant. The first pyloromyotomy was done on August 8, 1950, at five weeks of age. He was discharged August 19, gaining weight and retaining feedings. He did well for three weeks. Then he vomited all feedings, lost weight and was re-admitted for x-ray examination on September 18. The roentgen examination showed evidence of pyloric obstruction and on September 21 he was reoperated upon. Another site was chosen for the pyloromyotomy and the muscle fibers carefully divided. Following this second operative procedure, the infant has done well.

One of the most unusual complications, and one not previously described as a complication, although it is considered in the differential diagnosis of this condition, was an infant with chalasia of the esophagus. He also had a congenital hypertrophic pyloric stenosis. This infant was a premature male, born August 26, 1950, weighing 5 pounds at birth. He was admitted to the hospital September 28, because of vomiting since ten days of age. His weight on admission was 4 pounds 8 ounces, and he was severely dehydrated. He had visible gastric waves and a tumor mass was palpated. X-ray examination on October 7 showed pyloric obstruction, suggestive of congenital hypertrophic pyloric stenosis. A pyloromyotomy was done on October 10 and a good-sized tumor was found. He continued to regurgitate postoperatively and did not do well. On October 24 he was re-x-rayed, and the stomach was found to be emptying normally at this examination. On November 17, because of continuous vomiting, roentgen examination was again done and a diagnosis of chalasia of the esophagus made. As soon as postural treatment was instituted, this infant did well. He was discharged November 28, weighing 6 pounds 7 ounces. The survival of this infant was dependent on the excellent fluid therapy given. The value of Butler's solution in the fluid therapy was demonstrated in this case clinically and by laboratory findings in repeated instances.

Conclusion

Seventy-two infants had pyloromyotomies performed at the Children's Hospital, St. Paul, during the past six years.

The majority (87 per cent) of the infants treated were males.

Pylorospasm is one of the most difficult differential diagnosis to make in this disease. One of the infants in this group was operated upon before the diagnosis was made.

Preoperative and postoperative fluid therapy is the most important therapeutic consideration in reducing the mortality of this disease. The value of potassium in the fluid therapy of this condition was clinically evident.

The roentgen examination is not used frequently enough in the diagnosis of congenital hypertrophic pyloric stenosis.

Complications are not infrequent. The surgical complications encountered were: perforation of the duodenum, partial separation of the abdominal wound, hemorrhage, and one case requiring reoperation. The medical complications were pneumonia, otitis media, diarrhea, and chalasia of the esophagus.

Careful diagnosis and operation before severe dehydration are necessary if we are to improve the mortality of this disease.

The mortality rate, in this group, was one death in seventy-two cases, or 1.37 per cent. This compares favorably with any group of cases reported in the literature.

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Discussion

DR. ALEXANDER STEWART, Saint Paul: I just want to say that this is a very fine report on the modern treatment of pyloric stenosis. I have here a paper by Dr. Walter Ramsey written in 1906, forty-five years ago, on two cases of pyloric stenosis in which the condition was diagnosed and a gastroenterostomy performed, but both infants died.

Before the days of the Fredet-Ramstedt operation, according to Holt, the mortality rate was about 50 per cent in all cases of pyloric stenosis whether treated medically or surgically. Dr. Sedgwick originally felt that there was no such thing as pyloric stenosis and treated all his cases medically as pylorospasm; he had quite a large group and I think that eventually they all recovered.

Again, I wish to thank Dr. Flanagan for this fine study of our cases in the Children's Hospital and the report of a mortality rate of only 1.4 per cent.

DR. O. S. WYATT, Minneapolis: I think this is a fine review of what has gone on at the Children's Hospital in the care of pyloric stenosis cases for the past five years. Dr. Flanagan is quite right in going out of his way to compliment the nurses because I am sure that without good pediatric nurses we would still be losing some of these babies, particularly those who are brought in from the country and are not in too good shape. Regarding the palpation of the tumor, I think that in most instances if one is persistent he is able to pick up the tumor. Regarding pylorospasm, I must confess that I have opened up six or seven of these infants and much to my embarrassment have not found a pyloric tumor. Strangely enough, if you incise the serosa of the pylorus in exactly the same manner that you do for a Ramstedt operation, these babies stop vomiting at once and have a recovery just as smooth as though a Ramstedt had been done.

Regarding x-ray diagnosis, I do not believe that it is necessary except in the borderline cases and where we are not positive of the diagnosis. I have seen babies vomit the barium and then inhale it and get beautiful bronchograms. This we all know is not good for our babies and should not be used except under the above-mentioned conditions. Dr. Arey, who is here this evening, had the smallest baby we have ever done a Ramstedt on. This baby weighed 2 pounds 12 ounces at the time of operation and the tumor could be palpated. Incidentally, it was one of triplets.

I think the mortality rate at Children's Hospital is a commendable one.

I wish to thank Dr. Flanagan for the privilege of discussing his excellent paper.

DR. S. L. AREY, Minneapolis (by invitation): I enjoyed Dr. Flanagan's paper. I think the one most important thing is palpating the tumor, and I am sure all the pediatrists know the tricks but some of the general surgeons do not. If you will sit down and feed the baby, palpating the right upper quadrant just before or at the time the child vomits, you can feel the tumor.

DR. LOGAN LEVEN, Saint Paul: I enjoyed Dr. Flanagan's paper very much. It is of practical value and presents a definite regime for management of these cases that doctors in outlying districts will be able to follow. I am sure that the good results presented in this paper are a tribute to all concerned in the management of these cases.

While in many clinics these pyloric tumors apparently have been palpated in 100 per cent of cases, I still find it difficult to feel the pyloric tumor even under general anesthesia in many cases. In this series, only one infant was operated on that did not have a tumor at operation. In the majority of the cases a palpable tumor was not noted by the clinician preoperatively. Accordingly, it seems apparent that the criteria of a good history, visible peristalsis, and projectile vomiting are sufficient in most cases to make an accurate diagnosis of hypertrophic pyloric stenosis. I am rather opposed to the use of barium for x-ray study in any obstructive lesion of the upper gastrointestinal tract in children, due to the possibility of reguritation of barium and aspiration into the lungs. In most instances, the use of lipiodol as a contrast medium may be sufficient.

I am sure there may be a difference of opinion regarding the anesthetic of choice. Ether has been satisfactorily used in most of these cases. Likewise, all incisions have been right rectus, but I believe the merits of a transverse incision cannot be denied. When the pylorus is incised, there is always the danger of extending the

incision too far onto the duodenum. The chief danger in these cases is in not recognizing entrance into the duodenum at the time of operation. If this error in technique is recognized, the opening can be closed without danger.

During the postoperative period I am quite sure that good nursing care makes considerable difference in the feeding of these children.

This has been a very valuable presentation and the paper has practical application for all practitioners in the state.

DR. OWEN H. WANGENSTEEN, University of Minnesota: Dr. Flanagan alluded to the occasional occurrence in infants of chalasia (relaxation) of the esophagogastric juncture, a circumstance responsible for the occurrence of esophagitis in adults. He said nothing about the etiology of hypertrophic pyloric stenosis. I have observed pylorospasm of high grade in a male patient in his early thirties, whose primary disorder was mega-esophagus or cardiospasm. Neither the so-called cardiac sphincter of the esophagus nor the pyloric sphincter were hypertrophied; yet, the patient had a tremendously enlarged and tortuous esophagus and a very dilated stomach. I straightened out the redundancy in the esophagus by excising its distal 14 centimeters, together with the entire acid-secreting area of the stomach. This latter portion of the procedure precludes the occurrence of esophagitis, occasioned by the regurgitation of acid-peptic juice into the esophagus, a rather common sequel to the Heyrovsky procedure of esophago-gastric anastomosis for cardiospasm. The addition of a concomitant pyloroplasty in the case of the patient referred to above, rectified the associated pylorospasm.

Dr. Wyatt mentioned that he had seen infants with the clinical picture of hypertrophic pyloric stenosis, who presented no thickening of the pyloric sphincter, and that he performed myotomy upon the pylorus anyway. I would go along with that suggestion. In fact, I have come to think of mega-esophagus, hypertrophic pyloric stenosis and megacolon as being possibly variants of a somewhat related underlying disturbance. I have now operated upon seven patients with mega-esophagus in the manner outlined above, with very satisfactory results. None of them had a grossly detectable cardiac "tumor," such as is seen in hypertrophic pyloric stenosis. Nor do patients with megacolon have a "tumor" at the narrow constricted area in the upper rectum.

Patients with megacolon and mega-esophagus have one finding in common, viz: absence of nerve cells in Auerbach's plexus in the circular muscle. It would be interesting to know whether patients with hypertrophic pyloric stenosis exhibit this phenomenon. The stomachs of adult patients, who presented this syndrome and who have undergone gastric resection for the condition, might be studied to note whether Auerbach's plexus is abnormal, as it is in megacolon and mega-esophagus.

Hurst affixed the name of achalasia (failure of relaxation) to cardiospasm; I have come to speak of the condition as dystonia, because there is hypertonus of the distal esophageal segment and atony of the proximal reaches of the unstriated musculature of the esophagus.

Inasmuch as pylorotomy cures hypertrophic pyloric stenosis, and the Heller myotomy relieves the hypertonus of esophageal dystonia, is it likely that a similar procedure performed upon early Hirschsprung's megacolon might succeed in rectifying the situation? I think it is worth a trial in early cases.

DR. FLANAGAN (in closing): I want to thank the members for their discussions. I think there has been some work done on the relationship of Auerbach's plexus in the cause of pyloric stenosis. This is referred to by Szilagyi and McGraw: Problems of infantile pyloric stenosis with particular reference to surgical treatment. Surgery, 13:764-818 (May) 1943.

THE DOCTORS' INCOME

New light is cast on the earnings of doctors by a survey conducted jointly by the Department of Commerce and the American Medical Association. It shows that the average physician earns $11,058 yearly before income tax is paid. That doesn't, of course, include any unpaid bills on his books.

Because of a concentration of physicians in large cities, the doctor there earns, on the average, less than doctors in any other community size except villages of less than 2,500. Pennsylvania doctors averaged $10,047 and those in New Jersey $9,690. In New York the figure is even lower. Eight per cent of all the doctors made $25,000 or more, and 13 per cent had less than $3,000 a year net.

All of which contradicts a picture so frequently painted. Doctors with huge incomes seem the exception rather than the rule. Considering the fact that a doctor needs to spend at least four expensive and important years more in college than entrants into most other professions, the yield is not exorbitant. A bricklayer who put in as much overtime as most general practitioners would earn more.

The figures are important at this time, when a shortage of doctors is so much discussed. The incomes do a lot to explain the shortage.—Reproduced by courtesy of *The Sunday Bulletin,* Philadelphia, Pa.

AVASCULAR NECROSIS

Report of Cases Involving the Distal End of the Clavicle and the Odontoid Process

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RUMMEL, in 1895, wrote a classic monograph describing progressive compression of the vertebra following spine injury in which fracture was not primarily evident. His contentions have long been a matter for controversy, recently more generally accepted in the light of serial roentgenographic studies. Its occurrence is felt to be secondary to circulatory impairment following local thrombosis of the vertebral end arteries or nutrient artery.

Since Kummel's monograph, other lesions have been reported in which the question of vascular impairment as an etiological factor, in part or in whole, has been raised. The nature of the involvement of juvenile epiphyses and apophyses in such lesions has been the subject of much investigation as to whether it is infectious, neurogenic, metabolic or vascular. Osgood Schlatters' lesion of the adolescent tibial tubercle was described in 1903. Apophysitis of the juvenile os calcis was reported by Haglund in 1907. Osteochondritis deformans of the juvenile hip was described by Legg in 1910 and shortly thereafter by Perthe and Calve. In 1914 Freiberg reported osteochondritic involvement of the metatarsal head, and osteochondritis deformans juvenalis dorsi was classified as an epiphysitis by Scheuerman in 1921 with further reports by Calve and Buchman.

Another group of cases are clinically somewhat similar but involve adult bone suggesting more strongly impaired circulation as an etiological factor. Kohler's tarsal scaphoiditis, an aseptic osteitis (1908), Larson and Johansen disease of the patella (1921) previously described by Kohler (1908), Kienboch's disease of the carpal lunate (1910) and Presier's disease of the carpal scaphoid (1911) are classical examples of this group.

In 1900 Sudeck described the localized post-traumatic atrophy whose origin he thought infectious but later classified as a neuro-trophic or vasomotor atrophy. Post-traumatic atrophy of ordinary origin has been recognized since.

Tunner described bone atrophy with or without bone trauma but with definite neural injury in 1924. In 1925 Oserov (Esthonia) reported similar findings and in 1928 Ransahoff reported extensive osteoporosis of a fractured humerus in which the nutrient artery was injured. We now know that surgical trauma of bone, even mild, as in the removal of a small cortical graft from the tibia or a small osteoma from the femur may result in sufficient extensive atrophy to precipitate a pathological fracture.

Above are briefly mentioned the lesions which run the whole gamut of localized avascular necrosis or osteitis.

The classical avascular necrosis lesion, so familiar to many in late years, has been that of the head of the femur. This may occur from severe trauma, as fractures and dislocations, or mild trauma as from a single jarring blow.

Axhausen in 1928 attempted to explain these lesions on the basis of a minute embolus or thrombosis of the epiphysis or adjacent metaphysis. Phemister has done much to clarify the picture as a mild inflammatory or an embolic or thrombotic process due to a low-grade microorganism, with local necrosis followed by simultaneous absorption of dead bone and incomplete irregular replacement by new bone. Many disagree with the necessity for microorganisms, feeling that traumatic interference with the blood supply alone may initiate the process.

In 1938 I recognized avascular aseptic necrosis of the distal end of the clavicle for the first time. Thirteen other cases have since been recognized.

In 1946 our first of three cases of avascular atrophy of the odontoid process was observed.

Of these the following are typical.

Avascular Necrosis of the Distal End of the Clavicle

Case 1.—R.K. In October of 1937 this all-American football player was first seen having suffered a painful blow over the distal end of the clavicle. X-rays were negative. In January of 1938 I saw this patient again at which time he had local thickening of the outer clavicle, painful acromioclavicular motion. An x-ray showed a bulbous enlargement of the outer end of the clavicle, obvious clavicular cartilage base changes (subchondral) but a normal appearing acromial portion of the joint.

Presented before the Minnesota Academy of Medicine, March 14, 1951.

Case 2.—M.C. Early in November of 1939 this all-American injured his clavicle in similar fashion. By spring vacation, 1940, the acromioclavicular function was painfully and persistently impaired. The local and x-ray findings were similar to the preceding case. Excision of the clavicle distal to the coraco-clavicular ligament was carried out. The segment was grossly enlarged, and the clavicular cartilage was degenerated, while that of the acromium was normal in appearance. Microscopic examination showed a lesion similar to that found in aseptic necrosis of the head of the femur.

Case 3.—W.B. It has been my good fortune to have recently observed the development of this lesion over a period of months.

On February 20, 1947, this young man of seventeen years, captain of the Breck hockey team, was struck directly over the outer left clavicle with a hockey stick. On examination the joint was stable and not effused but there was local tenderness of the distal inch of the clavicle. X-rays on February 21 were negative. The patient was instructed to report at intervals for further x-rays on April 15, June 2, December 24, 1947, and March 3, 1948. During this period progressive periosteal and osseous bulbous enlargement occurred, and there was evidence of a degenerative subchondral type of irregularity of the distal clavicle without similar change on the acromial side of the joint. The patient stated that use of the shoulder produced such discomfort that he was limited in his athletic enthusiasm and that he would welcome surgery for relief. On June 21, 1948, excision of the clavicle distal to the coraco-clavicular ligament was carried out with relief of symptoms.

Case 4.—L.B. This twenty-eight-year-old patient was first seen by us on November 13, 1950, with a history of having sustained an acromio-clavciular separation on October 30, 1949. The shoulder was strapped for one week, and he was seen by three different doctors who told him the shoulder would take care of itself. In July, 1950, he started chiropractic treatments which consisted of pushing the clavicle downward once a week and associated heat treatments. After a period of three months of such treatment the soreness subsided and the patient was happy with the result. X-rays were taken and showed spontaneous absorption of the distal end of the clavicle.

I take no credit for the end result but I do feel the x-ray shows that avascular necrosis has occurred in the distal end of the clavicle.

Avascular Necrosis of the Odontoid Process

Case 5.—M.H. I first saw this patient on January 14, 1947. He was twenty-seven years of age. On August 13, 1946, he fell from a refrigerator car striking his head.

On August 14, 1946, an x-ray of the cervical spine showed a possible fracture line on the right side of the dens just lateral to the odontoid and extending into the base of the dens. There was no loss of alignment.

On August 17, repeat x-rays were negative. The above findings were confirmed by Dr. Rigler so the patient was discharged from the hospital on August 19. On August 26 the patient had developed severe pain in the neck so on August 30 he was readmitted to the St. Cloud Hospital. On September 7 the patient was fitted with a Thomas collar and was discharged on September 17, 1946. On October 25, 1946, he was quite comfortable so the collar was discontinued. He was considered well on November 25, 1946 and was to be discharged from care, but, with excellent judgment, his attending physician advised a final x-ray check.

The report follows: ". . . cervical spine now shows a definite irregular area of rarefaction extending from one side of the odontoid process to the other. The . . . process is moderately decalcified. It is now apparent that the line previously seen actually did represent a fracture line. No displacement. Marked decalcification about the fracture line."

The Thomas collar was reapplied.

On January 2, 1947, x-rays showed "slight further decalcification of the odontoid process."

On examination of the patient and on history there have been no neurological disturbances.

On January 14, 1947, x-rays by Dr. Malcolm Hanson confirmed the above reported findings.

On March 4, 1947, x-rays disclosed a moderate recalcification.

On May 1, 1947, there was further improvement.

On July 8, 1947, x-rays showed no essential change. The patient was seen at intervals and on December 7, 1948, x-rays were reported as follows: "Fracture involving the base of the odontoid process. There has been some absorption of bone at the fracture site with subsequent callus formation. The area of absorption has not been obliterated by callus."

I am indebted to Dr. J. A. Raetz of St. Cloud for his excellent co-operation in the follow up of this case.

These cases in my opinion add much to the support of the thesis that traumatic disturbance of the blood supply in the absence of infection may lead to avascular necrosis.

As far as I can find, this is the first report of avascular necrosis or atrophy in these locations.

Discussion

DR. EVANS (in closing): I would like to say just one thing and that is that if you look for this you will find it. These cases are not all surgical by any means. Out of fourteen cases, only six patients have had the distal end of the clavicle excised. I think this can occur from repeated trauma. One man was in a foundry and had to carry heavy loads on his shoulders.

DR. E. M. HAMMES, Saint Paul: What is the industrial attitude about these cases from the standpoint of compensation?

DR. EVANS: I have had three patients who were in industry and who had had persistent trouble. It really limits their effort of lifting, et cetera. They go on to what is a post-traumatic arthritis. The excision of the distal end of the clavicle will cure it. In none of the other cases have we had any disability; their shoulders are stable. We don't take enough of the clavicle out to reduce the stability.

PULMONARY TUBERCULOSIS AND CARCINOMA

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THE COEXISTENCE of pulmonary tuberculosis with malignant disease becomes commoner as the average age of the group in whom tuberculosis is found increases. Twenty years ago when this disease existed largely among adolescent and young adult groups, we rarely had the problem of treating conditions other than tuberculosis in the sanatorium. Today at Nopeming Sanatorium, while 57.5 per cent of the women patients are between twenty and thirty-five years of age, 58.5 per cent of the men are more than fifty years of age. It is in the latter group particularly that we are finding malignant disease, not only in other parts of the body but in the lungs as well.

Our experience with pulmonary carcinoma at Nopeming is weighted by the fact that, in addition to admitting patients with a preadmission diagnosis of tuberculosis, we also have accepted for diagnostic studies patients with pulmonary infiltrates of unknown etiology. A review of the incidence of malignant disease at Nopeming during 1948, 1949 and 1950 showed that eighteen patients with pulmonary tuberculosis also had malignant disease in other organs and eight had pulmonary tumors (seven bronchogenic carcinomas, one adenoma), for an incidence of combined disease in 3.1 per cent of the total admissions. In addition, pulmonary carcinoma was diagnosed in eighteen cases in which active tuberculosis was not demonstrated. With 1 exception all persons in whom carcinoma was demonstrated were more than fifty years of age and with four exceptions, all were men. A twenty-year-old Indian girl was found to have a bronchial adenoma together with pulmonary tuberculosis and a forty-seven-year-old woman had bronchial adenoma alone. A twenty-five-year-old tuberculous man was found to have a lymphoblastoma.

In our experience we have been impressed by the high incidence of all types of malignant lesions. The primary object of this paper, however, is to discuss the problems of pulmonary carcinoma and tuberculosis from the standpoint of diagnosis. Six patients with combined disease were treated in general hospitals for carcinoma before it was discovered that they had tuberculosis, and nurses and hospital personnel were unknowingly exposed to the major health hazard of this group. Had roentgenograms been taken as part of the routine of hospital admission this hazard would not have occurred.

In the group having combined pulmonary carcinoma and tuberculosis as well as in the group having pulmonary carcinoma alone, significant differences were not revealed by the histories. The symptoms of the entire group revealed the findings which are common to pulmonary lesions of this type. All had cough, either productive or nonproductive. Of this group, 70 per cent had lost from 5 to 30 pounds (2.3 to 13.6 kg.); 33 per cent had histories of hemoptysis. Many of those who had carcinoma alone gave histories of having had contact with tuberculosis. In spite of the fact that many of these patients had symptoms of more than a year's duration, in the majority of cases the pulmonary lesion was discovered by the Mobile Unit Chest Survey. The means of diagnosis for the eight patients having pulmonary neoplasm and tuberculosis are shown in Table I.

One patient had negative results from a Mantoux test in spite of the fact that tuberculosis developed in guinea pigs inoculated with gastric contents. In the patients who underwent operation tuberculosis was not reported by the pathologist. All resections were done elsewhere, and in two cases the results of gastric inoculations were not known until several weeks after operation had been performed. One patient underwent exploratory thoracotomy, at which time the preoperative diagnosis of carcinoma was confirmed by removal of a metastatic hilar node. In view of the fact that in all cases in which necropsy was performed both carcinoma and tuberculosis were demonstrated, the

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possibility of laboratory error in this group is doubtful, except possibly in Case 3, reported later in the paper, in which results of the tuberculin test were negative.

patient died of advanced pulmonary tuberculosis but necropsy revealed the presence of an unsuspected bronchogenic carcinoma. Table III does not include two cases, one case of lymphoblastoma associated with pulmonary tuberculosis and one case of bronchial adenoma alone. This latter case was of particular interest because of false-positive results of a sputum cytology test. One other instance of a false-positive result was encountered in our experience; the patient had a middle lobe syndrome. In the brief reports of cases which follow, roentgenograms were not necessarily characteristic of bronchogenic carcinoma and they did not always reveal evidence of combined carcinoma and tuberculosis.

TABLE I. MEANS OF DIAGNOSIS OF CARCINOMA AND PULMONARY TUBERCULOSIS

Case	Roentgenograms	Sputum		Culture or inoculation of guinea pigs	Biopsy of bronchial specimen	Operation	Necropsy	
		Carcinoma	Acid-fast bacilli				Carcinoma	Tuberculosis
1	Pos.	—	Pos.	Neg.	Neg.	—	Pos.	Pos.
2	Pos.	Pos.	Neg.	Pos.	Neg.	Pos.*		
3	Pos.	Neg.	Neg.	Pos.	Neg.	Pos.*		
4	Pos.	Neg.	Pos.	Pos.	Pos.†	Pos.		
5	Pos.	—	Pos.	Pos.	—	—	Pos.	Pos.
6	Pos.	Pos.	Pos.	Pos.	Neg.	—	Pos.	Pos.
7	Pos.	Neg.	Pos.	Neg.	—	Pos.*		
8	Pos.	—	Pos.	Pos.	Neg.	—	Pos.	Pos.

*Tuberculosis not reported.
†Bronchial adenoma.

TABLE II. BRONCHOGENIC CARCINOMA
Tuberculosis Not Demonstrated

Means of diagnosis	Number of cases	Results	
		Positive	Negative
Mantoux test	14	12	2
Carcinomatous cells in sputum	8	6	2
Acid-fast bacilli	16	0	16
Culture or inoculation of guinea pigs for tuberculosis	16	0	16
Biopsy of bronchial specimen	5	2	3
Biopsy (other)	2	2	0

TABLE III. PULMONARY TUBERCULOSIS AND MALIGNANT DISEASE
1948 through 1950

	Cases	Per cent
Pulmonary malignant disease	16	67
Pulmonary malignant disease and tuberculosis	8	33
	24*	100
Resection	7	29
Inoperable at thoracotomy	4	17
Medically inoperable	10	42
Operation not performed	2	8
Not diagnosed	1	4

*Total does not include one case of bronchial adenoma alone and one case of lymphoblastoma associated with pulmonary tuberculosis.

Means of diagnosis other than exploratory thoracotomy or necropsy in cases of bronchogenic carcinoma in which active tuberculosis was not demonstrated are shown in Table II.

The preponderance of positive results for Mantoux tests in this group is significant not only in the fact that this test is of no value in differential diagnosis but also in the fact that it demonstrates that most patients in the group in which carcinoma was proved have a tuberculous infection. When it is realized that tuberculosis may develop when an infected person becomes debilitated, it will be realized that in some cases superimposed active tuberculosis could have developed. The disposition of the cases of pulmonary malignant disease, with or without tuberculosis, is shown in Table III.

The study of sanatorium patients in this group averaged sixteen days. However, the average time before those patients who underwent resection were operated on was six weeks, because of indecision on the part of the patient and the fact that arrangements for operations had to be made elsewhere. One patient who was advised to undergo resection refused surgical intervention. Another

Report of Cases

Case 1.—A man, sixty-six years of age, was admitted to Nopeming Sanatorium February 1, 1950, with combined tuberculosis and bronchogenic carcinoma. Diagnosis was confirmed both by demonstration of tubercle bacilli in the sputum and gastric contents and by demonstration of neoplastic cells in the sputum and bronchial washings. He died February 22, 1951, and necropsy revealed far-advanced tuberculosis of the right upper lobe with cavitation and bronchogenic carcinoma in the same lobe.

Case 2.—A man, fifty-two years of age, was transferred to Nopeming Sanatorium as a candidate for tho-

racoplasty on March 22, 1950. Each month a specimen of sputum had been sent to the State Board of Health and each specimen was reported to have been positive up to the time of his transfer. On his admission at Nopeming Sanatorium physical examination revealed a tender swollen right arm, Horner's syndrome associated with ptosis of the right upper eyelid, and unilateral perspiration on the right side of his face. Tubercle bacilli were not demonstrated in tests at Nopeming. A diagnosis was made of probable pulmonary sulcus tumor (Pancoast), and after twenty-nine days arrangements were made to transfer him to the University Hospitals at Minneapolis. He died several days after extensive surgical measures and necropsy revealed carcinoma of the apex of the right lung involving the brachial plexus. Tuberculosis was not demonstrated by the pathologist who examined the specimen.

Case 3.—A woman, fifty-four years of age, was admitted to Nopeming Sanatorium September 13, 1950, as the result of a roentgenogram taken by a Mobile Unit and a diagnosis of pulmonary infiltration in the upper portion of the left lung. The diagnoses considered were pulmonary tuberculosis and carcinoma. Tubercle bacilli were not demonstrated and the result of the tuberculin test was negative. Several specimens of sputum failed to reveal definite presence of malignant cells though there was some question in the pathologist's mind. Because of a suggestion of bronchial occlusion and the inability to definitely rule out malignant disease, exploration was advised after six days' hospitalization. Cut section of a specimen removed for biopsy revealed an adenocarcinoma of the apex of the left lung. Several weeks after resection a guinea pig inoculated with gastric contents from the patient taken at the time of the sanatorium study was found to have tuberculosis. However, the surgical procedure, which to date has been successful, was not delayed by waiting for this laboratory data. Had we waited and received this report of positive results it is possible that the prolonged delay might have resulted in consideration that the patient had tuberculosis alone. Tuberculosis has not been demonstrated in the surgical specimen although the possibility of a laboratory error must be considered.

Case 4.—A woman, twenty-one years of age, was admitted to Nopeming Sanatorium March 28, 1950, with a proved diagnosis of pulmonary tuberculosis made earlier in another institution. She was transferred to Nopeming ostensibly for thoracoplasty. Examinations of sputum gave negative results for tuberculosis at the time of her admission but a guinea pig inoculated with gastric contents was found to show positive evidence of tuberculosis. Owing to findings at the hilus of the left lung, bronchoscopy was done previous to consideration of thoracoplasty. A pedunculated tumor was found obstructing the bronchus of the left upper lobe. Biopsy of this tumor revealed a bronchial adenoma. The patient was transferred elsewhere for resection which was accomplished, and the preoperative diagnosis of bronchial adenoma and tuberculosis was confirmed.

It is hoped that the presentation of this material will emphasize the fact that with the increasing age of persons having tuberculosis, particularly men, the incidence of combined tuberculosis and carcinoma is high; and further, that bronchogenic carcinoma and pulmonary tuberculosis can be coexistent. The danger of allowing a pulmonary malignant lesion to progress beyond the possibility of surgical intervention by suspecting tuberculosis rather than carcinoma as the cause of pulmonary infiltrations cannot be overemphasized. In the three cases in our series in which tuberculosis was demonstrated by inoculation of guinea pigs or culture, patients were sent for operation without awaiting results of laboratory findings. With the increasing safety of surgical procedures on the thorax, exploratory thoracotomy should be advised in all cases wherein bronchogenic carcinoma in an operable state is suspected. From the standpoint of public health it is hoped that our experience will impress the physicians with the fact that the demonstration of a malignant lesion in the lung or elsewhere does not rule out the possibility of a coexistent active tuberculosis which, if unknown, could result in innocent spread of this disease to hospital personnel and others associated with the patient. It is to be emphasized that debilitating conditions such as carcinoma can and often do cause latent tuberculosis to become active.

COUNTRY DOCTORS HAVE IT BETTER

Not the least of the satisfactions that compensate the physician who chooses to practice in a small city or town is a higher average income than his colleague in a large metropolitan center earns, according to cold figures compiled by the United States Department of Commerce. He not only has a larger dollar income, but his real income advantage is even greater, in view of the well known fact that living is more expensive in thickly congested urban areas. This inducement, plus the opportunity to be of service where they are most needed, should go far toward correcting the apparent shortage of doctors in rural communities.

The income survey, in which 55,000 physicians assisted, offers economic proof that a nation-wide shortage of physicians does not exist, as proponents of socialized medicine like to argue. Physicians' incomes have risen percentagewise in almost exact proportion to the average income of the American people, which suggests that supply and demand are rather nicely balanced. On the other hand, the income differential within the profession itself suggests there actually is an over-supply of physicians in the big cities. The significance of this fact will not escape the young medical graduate looking for a place to hang out his shingle.—*Indianapolis Star.*

CYTOLOGIC DIAGNOSIS OF UTERINE MALIGNANCIES

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THE purpose of this presentation is a practical evaluation of exfoliative cytologic diagnostic procedures for the detection of uterine malignancies. A by-product of the recent widespread interest in cytologic and biopsy studies of the cervix is a revival of a study of the earliest manifestations of carcinoma of the cervix.[49,53,54] Since it is generally agreed by some of the best authorities that these changes, namely, carcinoma *in situ*, are most easily detected by cytologic diagnostic procedures and since this malignancy and the cytologic means of recognizing it could be the best way of controlling cancer of the cervix, I shall first give you my impressions of intraepithelial malignancy of the cervix. This will be followed by a review of laboratory methods of diagnosis, indications for cytologic and biopsy techniques, common mistakes and economic considerations.

Carcinoma In Situ

Definition.—An *in situ* malignancy is a noninvading irreversible anaplasia of cells. It is entirely likely that there are two general types of *in situ* malignancy: those which have the capacity of immediate invasion and the group which cannot invade and yet have all the other histologic traits of malignancy. The latter seemingly must await further metabolic changes which will permit ingress to normal adjacent tissues. Both *in situ* lesions are the first established phase of most types of cancer. Frequently observed examples include lesions of the lips, skin, vulva, gastrointestinal tract, endometrium, cervix and breast (Figs. 1 to 5). This first phase is extremely transient in some malignancies and prolonged for many years in others. Theoretically the varieties found in a given type or location of a malignancy blend into each other. A focal area of *in situ* carcinoma is a commonly observed complication of benign papillary adenomas of the gastrointestinal tract. There is no sharply dividing line between the common benign keratosis following x-ray or sunlight exposure and an irreversible noninvading squamous cell malignancy. This probably also holds for the precursors of the common type of intraepithelial malignancy of the cervix. Until the lesion is well established, it may frequently appear in controversial transitional forms.

Clinical—Carcinoma *in situ* of the cervix is most often symptomless and exists in a grossly normal or only mildly eroded cervix.[8,13,16,18,21,25,43,46] Occasionally metrorrhagia or bleeding with trauma is the only indication of its presence.[48] Its frequency approximates one or two in 500 apparently normal women.[28,47,57] In some physicians' office practices, it may occur as often as one or two in 100 women patients.[13,24,25] A knowledge of the exact frequency must await additional reports. Although it has been found throughout womanhood, the average age is between thirty-five and forty years, while invading cancer of the cervix has an average age occurrence at approximately forty-eight years.[46] This indicates a possible average duration of the *in situ* state of from eight to ten years, a wide margin of time for control measures.

The proof of the inevitable invasive nature of carcinoma *in situ* is found in at least eighteen cases reported in the literature,[16,41,61] where the ingressing malignancy occurred as much as twelve years after a biopsy was taken from the intraepithelial malignancy. Of greater importance is the frequent occurrence of small early invading malignancies in typical relatively large *in situ* lesions and the frequent remnants of *in situ* lesions in even advanced malignancies of the cervix. The common observation of intraepithelial carcinoma of the lip and skin[5] reinforce the concept of the cervical analogue.

Morphologic Identity—As visualized by low power objective, the squamous epithelium of a part of the cervix is transformed into a very cellular and hyperchromatic layer of cells (Fig. 6).[31] On closer study, it is found made up of compact cells with large dark-staining nuclei and a relatively small amount of cytoplasm (Fig. 7). The normal orderly stratification of epithelial cells

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Fig. 1. Margin of carcinoma *in situ* of vulva with hyperkeratotic and otherwise normal epidermis to the right.

Fig. 2. Anaplastic gland to right of a near normal gland in the center. This is from the margin of an anaplastic non-invading lesion 1.5 cm. in diameter adjacent to a gastric ulcer.

Fig. 3. Preinvading squamous cell carcinoma of the lip on the basis of a keratosis.

Fig. 4. The lower glands are a part of a small area of malignant alteration of the large papilloma of the colon as seen in the seven upper glands. No invasive tendencies were found.

Fig. 5. Anaplasia in endometrial glands without demonstrable invasion.

in the stratum germinativim and the palisading of the basal layer is completely disrupted. A loss of polarity of nuclei is combined with considerable variation in size and shape of nuclei typical of squamous cell cancer. The same anaplastic changes frequently replace cervical glandular structures yet the basement membrane remains intact until true invasion occurs.

Fig. 6. Low power view of carcinoma *in situ* of the cervix with greatly increased cellularity, loss of orderly arrangement of cells, hyperchromatic staining of nuclei and great variation in size and shape of cells.

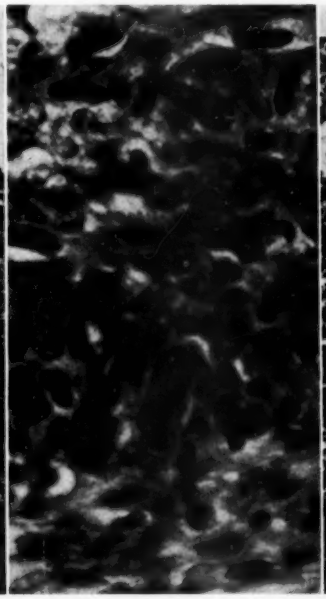

Fig. 7. High power view of Figure 6, lower margin of epidermis near the bottom of the photomicrograph.

Fig. 8. Early, small area of invading squamous cell carcinoma of cervix. Notice the near normal epidermis at top to right.

Classification—A review of our experience in 100 biopsies of invading carcinomas of the cervix (a poor source of material at best) reveals four possible types of origin of squamous cell malignancies. Each may be unicentric or multicentric. They include: (1) A frequent type of low grade slowly spreading (not invading) intraepithelial malignancy not necessarily preceded by leukoplakia or squamous cell mataplasia (Figs. 6 and 7). (2) A frequent rapidly invasive type of malignancy with only a transient *in situ* phase which is not likely to be found before invasion occurs (Fig. 8). Types 3 and 4 are less well defined. They are obvious sequelae of leukoplakia of the portio vaginalis and squamous cell metaplasia of the endocervix. Type 1 is the commonly known type of carcinoma *in situ*, representing one of the most widely discused, current gynecologic topics. In our study of cervical biopsies it might well have given rise to more than half of the squamous cell carcinomas of the cervix. The exact frequency of this type of onset of cervical cancer is of the greatest importance, and yet it remains to be determined. The invading offspring of this type of carcinoma *in situ* is frequently at high-grade malignancy which untreated will undoubtedly kill within a few years. Even after invasion starts there may well be a period of increasing growth momentum of an appreciable time.

Pregnancy—One of the most controversial and important present-day problems in obstetrics is the differential diagnosis of carcinoma *in situ* and reversible severely hyperplastic changes fairly frequently found in the cervices of pregnant women.[7,8,9,15,24,30] Many authors are now insisting that "carcinoma *in situ*" changes in the cervix disappear after the pregnancy; therefore, radical therapy is not indicated. Others perform hys-

terectomy or Wertheim procedures. Some are certain that they can differentiate cancerous from reversible hyperplastic histologic changes. This

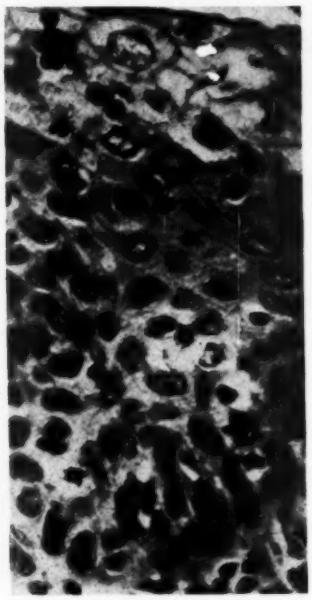

Fig. 9. High power view of hyperplastic cervical epidermis in pregnancy with loss of polarity of cells, also swelling, hyperchromatic staining and distortion of shapes of nuclei throughout the thickness of the epidermis.

new problem urgently needs clarification before a great deal more injury is perpetrated.

A nineteen-year-old pregnant woman developed a most severe cervicitis. The biopsy revealed a remarkable hyperplasia with loss of polarity in the basal cells, hyperchromatic staining of swollen nuclei, variation in size and shape of nuclei and numerous mitotic figures (Fig. 9). This patient was followed closely without radical therapy. Three years later, after having gone through two pregnancies, her cervix appears quite normal and smears do not contain cancer cells.

Treatment.—The procedures used for treatment of carcinoma *in situ* of the cervix include local resection, cautery, conization, trachelotomy, hysterectomy, pan-hysterectomy, radiation therapy and the Wertheim operation.[16,42,46,60] The diagnosis of carcinoma *in situ* is never established until the entire surface of the cervix and of the endocervix has been examined microscopically to reveal the absence of invasion in isolated or multicentric areas.[42] It is apparent that a minimum therapy is trachelotomy, so this fact can be determined. The specimen obtained by this procedure must be subjected to a careful histologic study as illustrated in Figs. 10 and 11. If no invading malignancy is found, the lesion is within the limits of the resected tissue following this serial block microscopic study and there is a good reason for preserving the uterus then, the therapy can be considered adequate. If not, a more extensive therapy is indicated. Where the facilities for this careful pathologic examination are not available, a minimum hysterectomy is indicated with possible radiation therapy alone or before and or after surgery. The extent of the malignancy in the hysterectomy specimen should be studied, contemplating postoperative radiation therapy.

Laboratory Methods of Diagnosis

Cytologic.—The principal cytologic techniques[3] of diagnosis of uterine neoplasms include: pipette aspiration or cotton tipped applicator collections of material from the vagina, cervix, cervical canal and endometrial cavity; also surface scrapings from the cervix obtained by dull applicators[3] and "sponge biopsy" procedures.[17] Direct smears are made of materials obtained by the above means on glass slides and placed immediately, before drying, in equal parts of ethel alcohol and ether or simply in 95 per cent alcohol. After an hour or more of fixation, these slides can be stained by one of several good methods or a drop of glycerine can be applied before covering with another slide and sending the preparation to a specialist for interpretation. The sponge biopsy and mucous secretions obtained can be studied by paraffin sectioning technique as well as by the smear method.

All of these procedures of obtaining cells for cytologic studies have proved successful in the hands of their advocates. Little can be gained by comparing the possible accuracy of the various methods. It has been repeatedly shown that combinations of the procedures are better than a single test. Thus it is not unusual for some authors to prepare smears from each, the vagina, cervix, and cervical canal. Obtaining material from the posterior cul-de-sac through aspiration pipette or on a swab which is protected in its passage through the vagina by plastic, rubber or

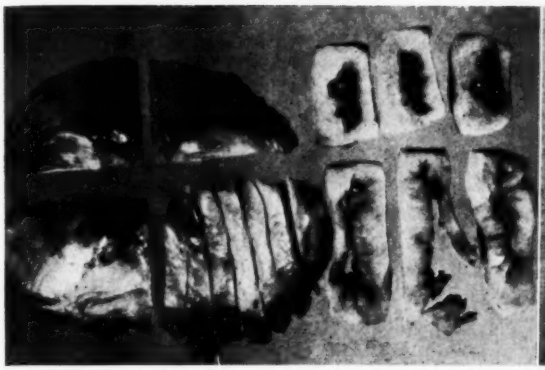

Fig. 10. The cervix is cut in quarters then sliced thin. The endocervix is cut horizontally.

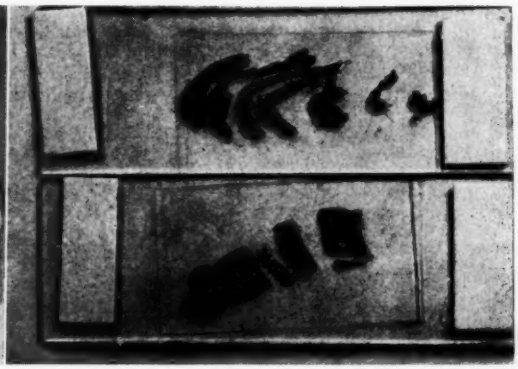

Fig. 11. The upper slide has all the blocks from one quarter of the cervix with deep connective tissues discarded. The lower slide has all the endocervix specimens.

heavy paper tubing is extremely economical in screening procedures involving large numbers of examinees where the service of a physician and direct visulization of the cervix are not feasible.[29] While smears obtained by rubbing the surface of the exposed cervix would seem to be more likely to obtain a higher concentration of cervical cancer cells than the diluted cul-de-sac collections of desquamated cells from the entire Muellerian tract.[3,13]

Mechanical screening devices are being developed by Papanicolaou and others in the hope that a high per cent of the negative smears can be discarded without wasting any time on these slides. This might be accomplished by the use of radioactive stains having an affinity for cancer cells or by other concentration and staining techniques.

Biopsy.—The widely used biopsy methods of diagnosing uterine malignancy include the use of: scalpel, cutting punches, sharp-edged scraping instruments,[41] electrocautery, conization procedures[23] and dilatation and curettage of the endometrium. It is apparent that cervical curetting or scraping procedures[41] intending to obtain thin intact sheets of epidermis from all parts of the cervix have a distinct place in cervices where there is no obvious site of cancer. This should be a routine procedure with every dilatation and curettage operation. The two specimens must be kept separate. Multiple biopsies are also useful in normal or near normal cervices.[51] It has been shown[12] that a biopsy of either the anterior or posterior leaflet alone will disclose only one half of the existing carcinomas *in situ* and approximately 80 per cent when both are taken. The addition of biopsies from the lateral aspects of the cervical os will disclose all but a few unusual carcinomas beginning high in the cervical canal.

Indications for Cytologic and Biopsy Techniques

Cytologic Criteria.—The method of diagnosis of malignancy through the study of small clumps of cells or single cells in smears or paraffin sections obtained from sputum, cavity fluid, gastric washings, vaginal or cervical exfoliations or urine, has beyond any question established itself as a useful procedure. The criteria of malignancy in the cells from these widely separated sources are essentially the same. The anaplastic intracellular traits are magnified in smear preparations over those found in solid tissue studies. This somewhat compensates for the failure to visualize any invasive tendencies, abnormal mitoses and altered polarity on the part of the cancer cells as best seen in biopsies. These deficiencies and the extreme sparsity of cancer cells in many "cytologic" technique preparations make the latter much more time consuming and difficult. In all such procedures negative findings do not rule out the presence of cancer, while the finding of cancer cells must be substantiated wherever possible by more dependable methods before radical therapy is instituted.

Generalizations.—A review of the more recent abundant literature on the subject of the diagnosis of uterine malignancies reveals a surprisingly

wide agreement among the authorities on a number of essential points.

1. The smear and biopsy are complementary procedures and should not be considered in conflict.[8,38,42,60] When carcinoma *in situ* lesions are included the combined accuracy of the two tests may be 98.3 per cent, whereas separate each may have a 10 per cent error.[1,19] The indications for both are fairly well defined.

2. Given an overt or "target lesion" always use the biopsy.[20,42]

3. Given a patient with intermenstrual or post menopausal bleeding of traumatic or spontaneous origin or persistent unexplainable discharge always do a biopsy and/or a dilatation and curettage. A supplementary smear study in this instance would be a good practice.[14]

4. The smear diagnosis of malignancy must always be substantiated by biopsy and/or dilatation and curettage, before definitive therapy is instituted.[26,29,38,39,51]

5. The most competent examiners find the smear method more accurate than a single biopsy in the detection of carcinoma *in situ* in normal appearing cervices.[24,41,42,54,57,58]

6. In the near normal cervix properly preformed epidermal curetting procedures using a sharp margined spoon,[41] scalpel or curette could surpass cytologic methods in accuracy. Only a slight oozing of blood is to be expected whereas multiple biopsies may require hospitalization. A wide application of the procedure is indicated.

7. The smear procedures are most useful in the screening of large numbers of apparently healthy women for cancer.[4,29,39,41,42]

8. In the hands of experts, false negative smears for carcinoma of the fundus approximate between 10 and 20 per cent. Some pathologists will verbally admit a 50 per cent error.

9. Because of this large possible error and since all but a very few cases of cancer of the fundus give sufficiently early clinical evidence of their presence, cytologic diagnostic procedures should be aimed primarily at detecting cancer *in situ* of the cervix and not fundal carcinoma.

10. The division of cells found in smear examinations into several degrees of "precancerous changes" is not warranted.

11. Smears are generally considered useful in follow-up of radiation therapy.[14,50,51]

12. Patients with repeatedly positive smears must be followed with the greatest care in spite of repeatedly negative cervical and endometrial biopsies.[20]

Common Mistakes

Common present-day mistakes include: (1) The widespread belief that all cancer of the cervix can be seen as an irregularity, ulceration or tumor mass; (2) the infrequency of taking multiple biopsies or epidermal thick scrapings from the cervix; (3) the failure to take cervical smears in patients who want and can afford this protection against cancer; (4) the failure to obtain biopsies of the cervix and endometrium and possibly cytologic smears from all patients with intermenstrual or post menopausal bleeding or unexplained discharge; (5) the frequency with which the cervical biopsy is mutulated and much of the epithelial surface is destroyed particularly when the electric cutting instruments are used; (6) many biopsies do not include the squamocolumnar junction and generally should be considered inadequate; (7) failure to obtain good epidermis, thin scrapings or multiple biopsies from the cervix separably from the portio vaginalis and endocervix routinely with every "dilatation and curettage" operation; (8) the idea that biopsies are infallible; (9) failure to obtain multiple biopsies preceding cautery therapy of the cervix; (10) the belief that cytologic diagnoses are a physician's office procedure. Years of disciplining in microscopic diagnostic methods, the experience of many false reports and the determined patience of a brooding hen are necessary for success.

Economic Considerations

The ultimate goal in the use of cytologic procedures for the diagnosis of uterine malignancies is sometimes considered to be the saving of the lives of over 2 per cent of all women over thirty years of age now living, who would eventually die of these malignancies. According to the National Office of Vital Statistics 17,120 women die in the United States in a single year from cancer of the uterus or 0.395 per 100 female population annually or 2.7 per 100 female deaths from all causes during that year.[55]

Consider it true that the great majority of cancers of the cervix are proceded by an *in situ* malignancy of at least five years' duration and that a cervical smear taken from each woman over thirty years every five years, would protect against

invading cancer of the cervix. Theoretically 1.5 women in every 100 now living in this age group could be saved from death of cancer of the cervix at the cost of one such periodic examination every five years. If this was a part of a general periodic health examination, the cost of visualization of the cervix might be prorated as low as one dollar. With volume work and trained screening assistants a laboratory fee of $3.00 per case may be feasible. Under these ideal circumstances, it would cost roughly $267 to save one woman from death by cancer of the cervix. This, then, might be considered a lifetime insurance premium against death by cancer of the cervix. If experience reveals that annual examinations are necessary the lifetime premium is $1,335. These figures would not include the cost of the patient's time and other expense or the repeated examinations and hospitalizations resulting from the false "suspicious cells" findings. The latter have run as high as 18 per cent in some reports.[50] Physicians submitting cervical smears annually from entirely normal women over thirty years of age should expect 333 negative tests for every positive test.

If we can believe the frequency of one or two cases of cancer *in situ* of the cervix per 100 women reported from physicians' office practices,[13,24,25] then there should be no question about the economic advisability of protection by cytologic smear studies in this group of women. A lifetime insurance premium here would cost $200. It is impossible today to do more than cite widely varied figures on the frequency of carcinoma *in situ* of the cervix in different selected groups and speculate on their accuracy.

At the present rate of interest and investigation, we should have within the next ten years much more accurate information upon which to estimate the cost per life saved in specified selected groups of women through cytologic examinations. This same research is being carried out in all fields of periodic examinations for cancer. There is no question but that much good is being done in cancer detection centers. The question is how many lives are saved beyond those saved without the aid of detection centers and the cost of each life saved in this group.

Conclusions

1. Cytologic smears are used primarily for the purpose of detecting carcinoma *in situ* of the cervix before a clinically apparent neoplasm has developed.

2. Carcinoma *in situ* of the cervix can vary from a rapidly transient lesion to one unable to invade for many years.

3. Upon the frequency of the slow variety of carcinoma *in situ* of the cervix depends the practicality of cytologic diagnostic procedures in this area.

4. If the great majority of cancers of the cervix are preceded by the five-to ten-year type of carcinoma *in situ* and if it is possible to train sufficient and accurate technical personnel for the purpose, it is conceivable that a thirty-year or older woman could insure herself against a 1.5 per cent chance of death by cancer of the cervix for a lifetime premium of $267.00.

5. There now exists in private practice a necessity of an almost universal improvement in surgical technique in connection with the diagnosis of carcinoma *in situ* of the cervix.

6. A clarification of the histologic difference between reversible hyperplastic cellular changes in the cervix of pregnancy and a true carcinoma *in situ* is urgently needed.

7. The criteria for the use of cytologic and biopsy techniques for detection of uterine malignancies are now fairly clearly defined.

8. In my opinion, it is only a matter of time before the general economic level and the customs of the people will lead to a wide use of cytologic or biopsy diagnostic procedures for the detection of cervical malignancy. These will be performed primarily in conjunction with periodic examinations for cancer on apparently healthy women by the general practitioner.

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ACCIDENTAL DEATHS IN MINNESOTA IN 1950

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MORE children are killed by accidents than by any other cause. In 1950, 190 Minnesota children from one to fifteen years of age were killed by accidents. This total represented one-third of all the deaths in this age period (Fig. 1). deaths from accidents has occurred during this same period.

Even during infancy, when the baby is entirely dependent upon an adult for his care, his health, and his safety, there were forty-four accidental

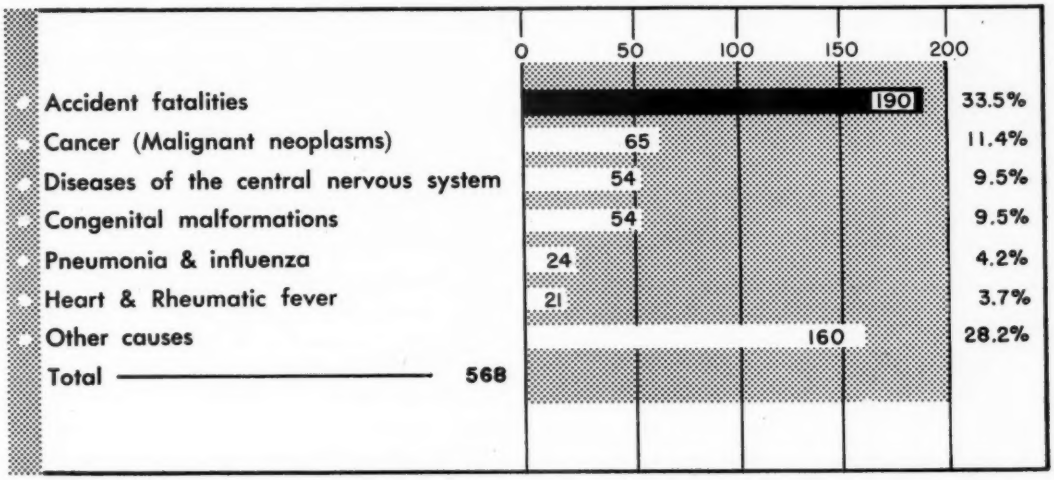

Fig. 1. Deaths from selected causes, ages one to fourteen years, in Minnesota residents, 1950.

Almost one-third of all the accidental deaths were motor vehicle fatalities. There were almost as many deaths from accidents as from malignant neoplasms, convulsions and diseases of the central nervous system, congenital malformations, pneumonia and influenza combined. These conditions caused a total of 197 deaths.

In recent years the reduction in deaths from disease has been more than twice as great as the reduction in deaths from accidents. The effects of preventive medicine and public health and the advances in clinical medicine have been noteworthy in reducing deaths from infectious and communicable disease during childhood. A decrease of 65 per cent has occurred in these deaths during the past thirty-five years. Accidental deaths, however, have assumed a more prominent place because only a 30 per cent reduction in deaths, in most cases due to someone's carelessness. It is true that this number is small in comparison with the total number of infant deaths (1,876) which are due chiefly to immaturity, congenital malformations, and birth injuries. It is important to note, however, that while there has been a 60 per cent decrease in infant deaths from all causes from 1915 to 1950, there has been only a 10 per cent decrease in deaths due to accidents during the same period. Mechanical suffocation in bed or cradle was reported in two-fifths of the accidental deaths in infants (Table IV.) While it is now generally accepted that deaths of infants ascribed to mechanical suffocation are, for the most part, actually the result of an acute respiratory infection, physicians and coroners continue to report these deaths as accidental. Burns were the second most frequent cause of accidental deaths in infants, with obstruction, suffocation or puncture by ingested objects in third place.

In the age group of one to five years, acci-

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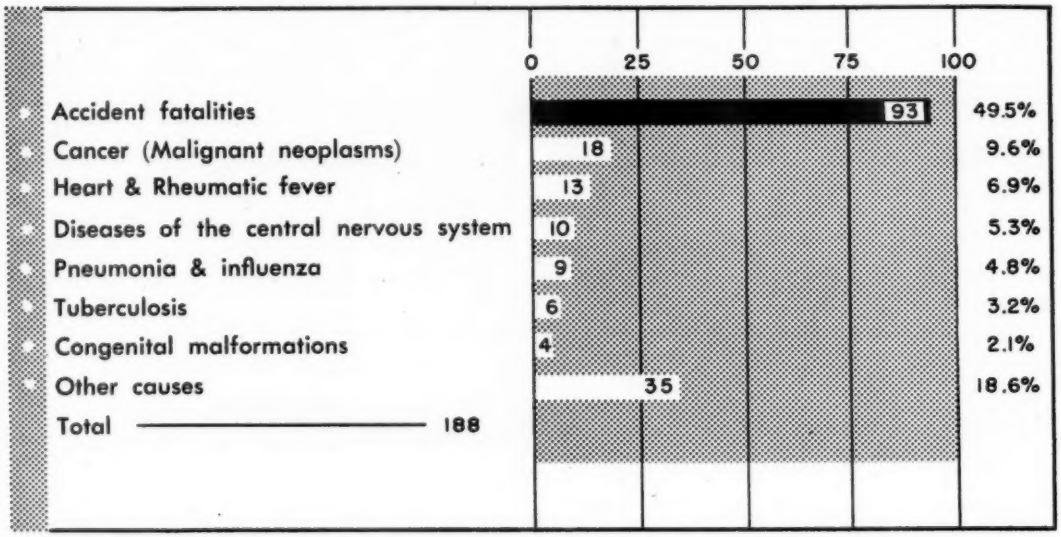

Fig. 2. Deaths from selected causes, ages fifteen to nineteen years, in Minnesota residents, 1950.

dental deaths constituted 28 per cent of all deaths; in the five to ten year age group, 36.6 per cent and in the age group of ten to fifteen years, accidents were responsible for 44 per cent of the deaths.

Among young people between the ages of fifteen and nineteen, almost one-half of all the deaths (49.5 per cent) were due to accidents (Fig. 2). More than half of these deaths were caused by motor vehicle accidents. These constituted 27 per cent of all the deaths. In contrast, deaths from malignant neoplasms, convulsions and diseases of the central nervous system, rheumatic fever, heart disease, pneumonia, influenza, tuberculosis, and congenital malformations constituted only 32 per cent of the deaths in this age period.

In young adulthood, between the ages of twenty and thirty-four years, accidental deaths continued in first place and constituted 36 per cent of the deaths (Fig. 3). More than half of these deaths were motor vehicle fatalities, which constituted 57 per cent of all the accidental deaths. Accidental deaths in this age group exceeded the combined deaths from malignant neoplasms, heart disease, tuberculosis, nephritis and nephrosis, diabetes mellitus, and maternal causes.

In the age group of thirty-five to sixty-four years, heart disease, cancer, and intracranial vascular lesions, the degenerative diseases most commonly associated with old age, were the three leading causes of death, with accidental fatalities in fourth place (Table I). Accidental deaths exceeded the combined total of all deaths from tuberculosis, pneumonia and influenza, and diabetes.

In the age group of sixty-five years and over, general arteriosclerosis, another of the degenerative diseases of old age, took fourth place and accidental deaths dropped to fifth place. But accidental deaths exceeded the number of deaths from pneumonia and influenza (Table II).

These figures show that accidents were not only the greatest health hazard for children but also were the leading cause of death up to age thirty-five, beyond which the degenerative diseases took the lead. The importance of accidents as a cause of death is emphasized even more when the ten leading causes of death are considered (Fig. 4). For all ages and all causes in 1950, accidental deaths were in fourth place, exceeded only by the leading degenerative diseases (heart disease, cancer, and intracranial vascular lesions). Motor vehicle accidents were responsible for one-third of all accidental fatalities (33.4 per cent). Accidental deaths in Minnesota residents (1,747) exceeded the total caused by pneumonia and influenza (897) and general arteriosclerosis (822).

Dickenson and Welker of the Bureau of Medical Economic Research of the American Medical Association, in a bulletin entitled "What is the Leading Cause of Death?" (1948), have proposed two new measures of the loss to society re-

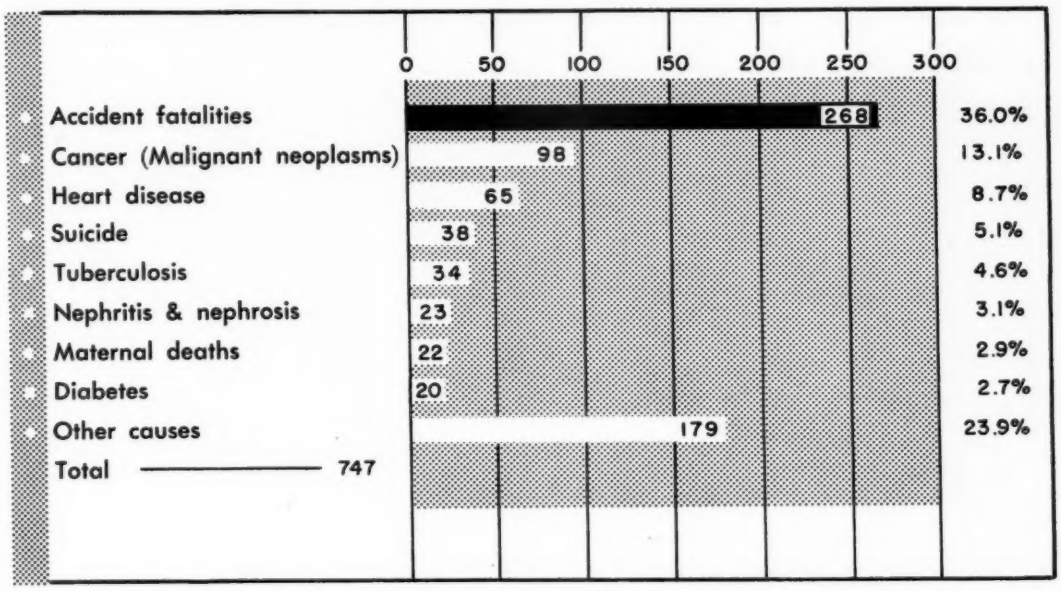

Fig. 3. Deaths from selected causes, ages twenty to thirty-four years, in Minnesota residents, 1950.

TABLE I. DEATHS FROM SELECTED CAUSES, AGES 35-64 YEARS, MINNESOTA RESIDENTS, 1950

Total (All Causes)	Number	Per Cent
	7,617	100.0
1. Heart disease	2,894	38.0
2. Malignant neoplasms	1,686	22.1
3. Intracranial vascular lesions	706	9.3
4. Accident fatalities	503	6.6
5. Suicide	234	3.1
6. Tuberculosis	171	2.2
7. Pneumonia-influenza	123	1.6
8. Diabetes	119	1.6
9. Nephritis and nephrosis	108	1.4
All other causes	1,073	14.1

TABLE II. DEATHS FROM SELECTED CAUSES, AGES 65 YEARS AND OVER, MINNESOTA RESIDENTS, 1950

Total (All Causes)	Number	Per Cent
	16,901	100.0
1. Heart disease	7,214	42.7
2. Intracranial vascular lesions	2,750	16.3
3. Malignant neoplasms	2,501	14.8
4. General arteriosclerosis	796	4.7
5. Accident fatalities	648	3.8
6. Pneumonia-influenza	565	3.3
7. Diabetes mellitus	381	2.3
8. Hypertension without mention of heart disease	222	1.3
9. Nephritis and nephrosis	124	0.7
All other causes	1,700	10.1

sulting from the leading causes of death, (1) "live years lost" and (2) "working years lost." They have defined "working years lost" as the average number of years to be lived between the twentieth and sixty-fifth birthdays, because this period is the earning period of life according to the usual working habits of persons in this country. From a study of the leading causes of death in the United States in 1945, they found that, while accidents were the fourth leading cause of death, they were the leading cause of working years lost, responsible for 12.6 per cent of all lost working years. Heart disease was the leading cause in number of deaths, but was in second place (12.2 per cent) for lost working years. Pneumonia, in sixth place in the United States as a cause of death, was the third leading cause for lost working years (8 per cent); cancer, in second place as a cause of death, was in fourth place in causing lost working years (7.5 per cent); intracranial vascular lesions, in third place as a cause of death, dropped to sixth place as a cause of lost working years (3 per cent).

Accidental Death Summary

Since 1938 there has been an average of 1,879 fatal accidents every year in Minnesota. Accidental death rates have declined from 69 per 100,000 population in 1938 (1,895 deaths) to 57.4 in 1950 (1,712 deaths), a decrease of 17 per cent (Fig. 5). There were two peaks, a rise in the rate to 75.8 per 100,000 population in 1941 and to 72.0 in 1945, at the beginning and at the end of the war years. The trend in motor vehicle

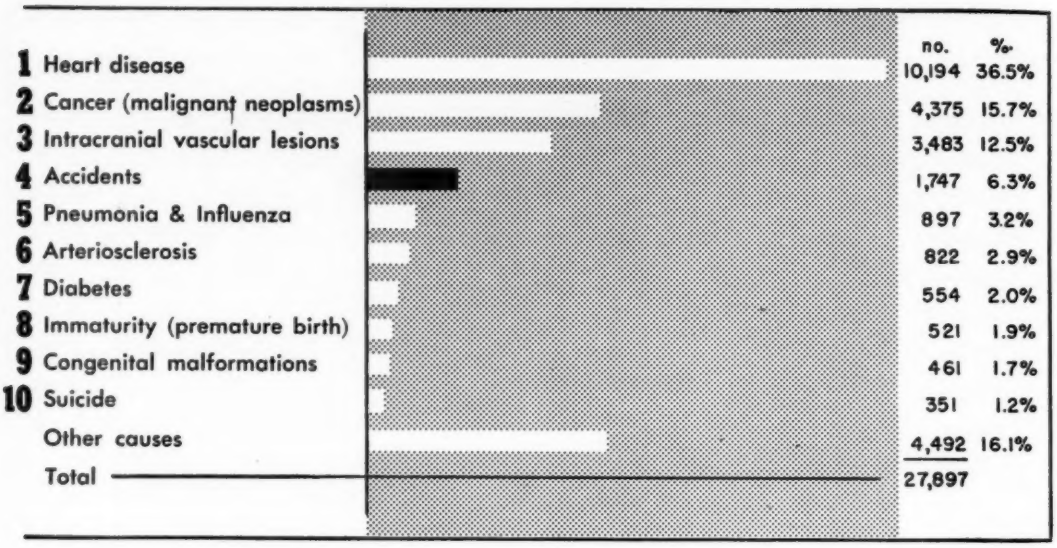

Fig. 4. Ten leading causes of death in Minnesota residents, 1950.

TABLE III. ACCIDENTAL DEATH SUMMARY, BY OCCURRENCE, MINNESOTA, 1950

Type of Accident	All Ages	Sex		0-4 Years	5-14 Years	15-24 Years	25-44 Years	45-64 Years	65 and Over
		M.	F.						
Grand Total	1,712	1,165	547	132	102	195	299	340	644
Home	660	315	345	90	25	14	43	74	414
Motor vehicle	559	431	128	27	31	118	133	140	110
Public	349	277	72	15	40	48	83	72	91
Occupational total	144	142	2	0	6	15	40	54	29

deaths during the past decade is of interest. There was a marked decrease during the war years (1941-1945) with a low of 11.8 per 100,000 population in 1943, due to the limitation in the use of motor vehicles. The death toll in the post-war years has not reached the high level of the prewar years despite the fact that highway travel has reached an all-time high in recent years. Coincident with the decrease in motor vehicle fatalities there was a rise in home, public, and occupational accidents, the latter rise doubtless being due to the tremendous increase in employment.*

A total of 1,712 accidental deaths occurred in Minnesota in 1950, a decrease of 10 per cent from the 1949 total of 1,904 deaths (Table III). This apparent decrease, however, was not actually due to a decrease in accidental deaths but to a change in statistical coding, which took place in 1950.

*Provisional figures to date for 1951, however, indicate that if motor vehicle deaths continue at the current trend the highway accident death toll may exceed that of any previous year.

Twice as many accidental deaths occurred in males as in females. Two out of five accidental deaths were home accidents (38.6 per cent). Only in this type of accident were there more female than male accidental deaths (345 female; 315 male). Out of a total of 660 home accidental deaths, ninety were in persons under the age of five years and 414 in persons sixty-five and over. One out of every three were motor vehicle deaths (33.3 per cent) and one out of five were public non-motor vehicle deaths (19.7 per cent). One out of twelve were occupational accidents (8.4 per cent) and one-half of these were accidental deaths occurring in agriculture (Fig. 6).

By specific age groups (Table III), in children under the age of five years, 68 per cent (ninety deaths) were home accidents, 21 per cent (twenty-seven deaths) were motor vehicle fatalities, and 11 per cent (fifteen deaths) were other public accidents. In school children between the ages of five and fourteen years, home fatalities dropped to

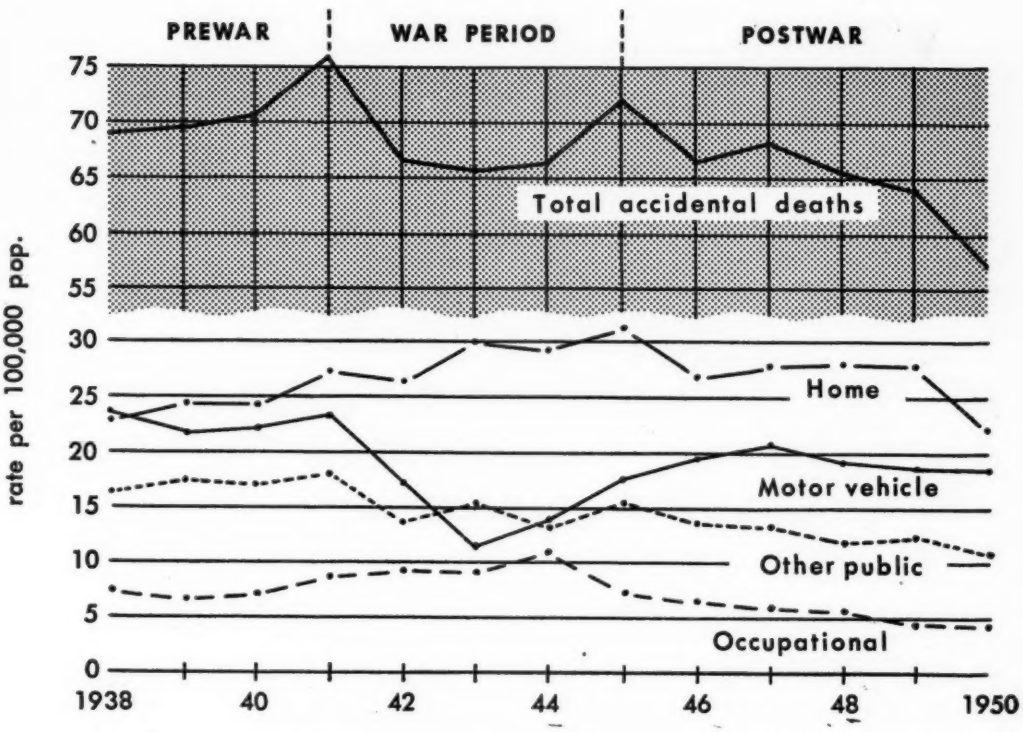

Fig. 5. Accident mortality rates by major classification, by occurrence, in Minnesota, 1938-1950.

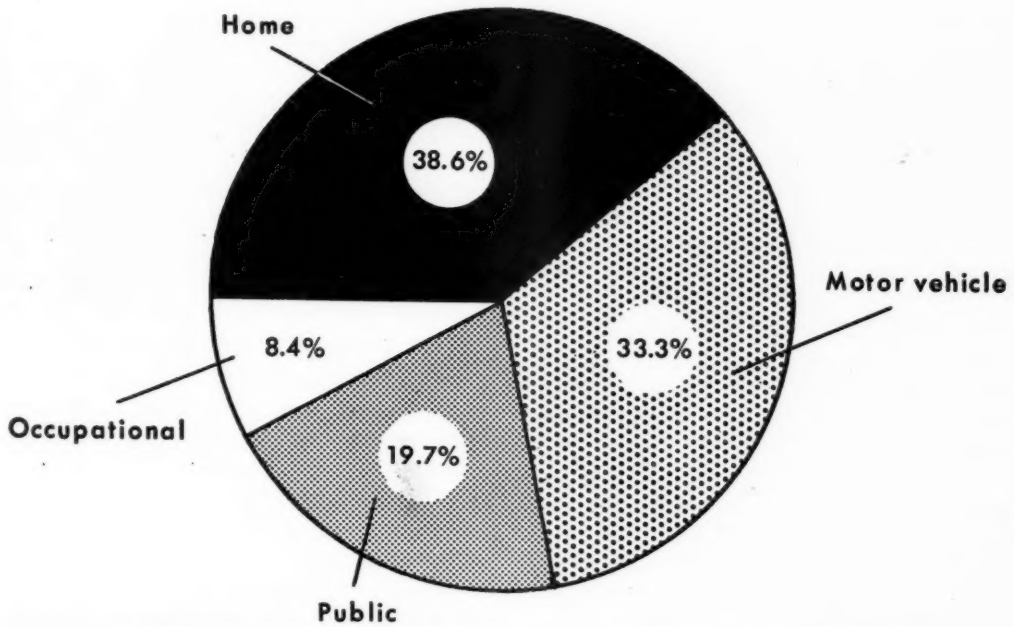

Fig. 6. Accidental deaths by major classification by percentage, by occurrence, in Minnesota, 1950.

TABLE IV. PRINCIPAL CAUSES OF HOME ACCIDENT FATALITIES—
NUMBER AND PER CENT DISTRIBUTION FOR SELECTED AGE GROUPS, BY OCCURRENCE, MINNESOTA, 1950

Type of Accident	All Ages		Under One Year		1-4 Years		5-14 Years		15-24 Years		25-44 Years		45-64 Years		65 & Over	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Total	660	100.0	44	100.0	56	100.0	25	100.0	14	100.0	43	100.0	74	100.0	414	100.0
Falls	426	65.0	2	4.6	2	3.6	3	12.0	—	—	9	21.0	39	53.0	371	89.6
Burns	94	13.7	8	18.0	20	35.7	11	44.0	2	14.3	16	37.2	14	18.9	23	5.6
Mechanical suffocation	19	2.9	18	40.9	1	1.8	—	—	—	—	—	—	—	—	—	—
Poisonings by gases	17	2.6	—	—	—	—	—	—	1	7.1	5	11.6	6	8.0	5	1.2
Firearms	16	2.4	2	4.6	—	—	1	4.0	7	50.0	3	7.0	3	4.0	1	0.2
Poisonings (gas excepted)	15	2.3	2	4.6	4	7.1	—	—	—	—	5	11.6	3	4.0	1	0.2
Drowning	14	2.1	1	2.3	12	21.4	1	4.0	—	—	—	—	—	—	—	—
Suffocation by foreign objects	11	1.7	4	9.1	3	5.4	1	4.0	—	—	1	2.3	1	1.3	1	0.2
Injury by farm machinery	8	1.2	—	—	5	8.9	2	8.0	—	—	1	2.3	—	—	—	—
Other	40	6.1	7	15.9	9	16.1	6	24.0	4	28.6	3	7.0	8	10.8	12	2.9

TABLE V. MOTOR VEHICLE DEATHS, BY OCCURRENCE, MINNESOTA, 1950

Cause	All Ages	Per Cent of Total	0-4 Years	5-14 Years	15-24 Years	25-44 Years	45-64 Years	65 & Over
Total	559	100.0	27	31	118	133	140	110
Collision with other motor vehicle	179	32.0	3	3	42	45	62	24
Injury to pedestrians	154	27.6	18	15	13	11	35	62
Non-collision	134	24.0	4	3	40	51	24	12
Collision with railroad train	46	8.2	2	3	11	13	10	7
Collision with fixed object	31	5.5	—	—	10	11	8	2
Collision with bicycle	10	1.8	—	7	1	—	1	1
Other	5	0.9	—	—	1	2	—	2

25 per cent (twenty-five deaths) while motor vehicle deaths increased to 31 per cent (thirty-two deaths) and public accidents to 38 per cent (thirty-nine deaths). In young people between fifteen and twenty-four years, motor vehicle fatalities doubled to 61 per cent (119 deaths) while home accidents decreased to 7 per cent (fourteen deaths). Between the ages of twenty-five and forty-four years, motor vehicle fatalities were responsible for 43 per cent of the accidental deaths (135 deaths) and home fatalities accounted for 18 per cent (forty-three deaths). In the age group sixty-five and over, home accidents accounted for 414 deaths or 64 per cent of all accidental deaths, and motor vehicle fatalities totaled 115 deaths or 18 per cent.

Home Accidents

Home accidents are of special interest, not only because they constituted two-fifths of all accidental deaths, but because one-half of all accidental deaths in children under the age of fifteen were home accidents. The principal cause of home accidents was falls, which were responsible for more than two-thirds of all home accidental deaths (sixty-five per cent) (Table IV). Fatal falls occurred in older persons, 9.5 per cent of all such deaths occurring in persons between the ages of forty-five and sixty-four, and eighty-seven per cent in persons who were sixty-five years of age and older. The second cause of accidental deaths was burns, responsible for fourteen per cent of the deaths from home accidents. Almost one-fourth of the deaths from burns occurred in children under age five and another one-fourth in persons sixty-five years of age and older. More than one-third of all accidental deaths in children between the ages of one and five years were due to burns (36 per cent). Two-fifths of the poisonings from solids or liquids occurred in children under age five, while poisonings from gases and vapors occurred in adults. Almost one-half of the home deaths from firearms occurred in persons from fifteen to twenty-four years of age. Almost all of the home drownings occurred in children under the age of five years (93 per cent). It should be noted that home drownings represent only a small portion of the total number of drownings in Minnesota. Most of them are tabulated under public accidents (Table VII), where additional deaths from firearms will be found.

Motor Vehicle Accidents

In 1950 there were 559 fatal motor vehicle accidents, the same as in 1949, this number comprising 33 per cent of all fatal accidents. Collision

with another motor vehicle caused 32 per cent of the deaths; injury to pedestrians caused 28 per cent of the fatalities; and non-collision accidents were responsible for 24 per cent. Five per cent per cent (110 deaths) at age sixty-five and over. In the oldest age group, as in children, pedestrian injuries constituted more than one-half of the motor vehicle deaths.

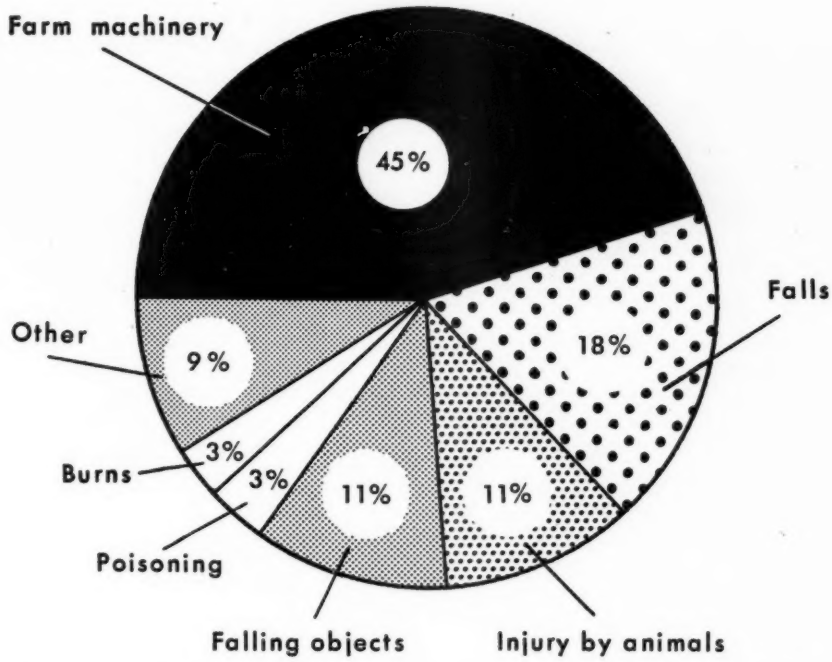

Fig. 7. Farm fatalities by specific cause and percentage, by occurrence. in Minnesota, 1950

TABLE VI. OCCUPATIONAL ACCIDENTAL DEATHS, BY OCCURRENCE, MINNESOTA, 1950

Type of Accident	All Ages	Per Cent of Total	0-4 Years	5-14 Years	15-24 Years	25-44 Years	45-64 Years	65 & Over
Total	144	100.0	—	6	15	40	54	29
Agriculture	73	50.7	—	6	9	13	26	19
Manufacturing	11	7.6	—	—	3	4	4	—
Transportation	10	6.9	—	—	—	2	4	4
Mining, quarrying, oil and gas wells	9	6.2	—	—	1	4	3	1
Public utilities	8	5.6	—	—	1	2	3	2
Construction	7	4.9	—	—	1	3	3	—
Other	26	18.1	—	—	—	12	11	3

(twenty-seven deaths) of motor vehicle deaths occurred in children under age five, two thirds of them as pedestrian injuries; another 5 per cent (thirty-one deaths) between the ages of five and fourteen years, one-half being pedestrian injuries; 21 per cent (118 deaths) of motor vehicle deaths occurred between fifteen and twenty-four years; 24 per cent (133 deaths) between twenty-five and forty-four years; 25 per cent (140 deaths) between forty-five and sixty-four years, and twenty

Occupational Accidents

There were 144 occupational deaths in 1950 representing 8.4 per cent of all accidental fatalities in Minnesota (Table VI). Farm fatalities totaled seventy-three, more than half of all occupational deaths (50.7 per cent). Farm machinery was the leading farm hazard, accounting for 45 per cent of the farm deaths. Falls were responsible for 18 per cent; injury by animals and falling objects accounted for 11 per cent each; burns and poison-

ing accounted for 3 per cent each (Fig. 7). Only six deaths from occupational causes occurred between the ages of five and fourteen years. These were all farm accidents, and they constituted 8 per cent of all farm accident deaths.

Summary

Accidents are the greatest hazard in the lives of children. Although accidents are responsible for only 3 per cent of the deaths under the age of one year, they are responsible for one-third of all the deaths between the ages of one and fifteen years, and one-half of them are home fatalities. Accidents are the cause of one-half of the deaths between the ages of fifteen and nineteen years. They continue as the leading cause of death up to age thirty-five, beyond which the degenerative diseases take the lead. Among the ten leading causes of death, accidents continue to hold fourth place in number of deaths. But, quoting Dickenson and Welker, "We see that fatal accidents outrank every other cause of death as a destroyer of the working years of life, the years which represent the productive and military strength of this country."

Statistics on accident fatalities tell only part of the story. Many more children and adults are handicapped and disabled by non-fatal accidents, which are not reportable in Minnesota. Most accidental deaths are preventable. We need a greater appreciation of the size of the problem and more knowledge on how and why accidents occur. The medical profession, health departments, schools, welfare agencies, and safety groups are all interested in child and adult health, safety and welfare. Accident prevention is a tremendous job that requires the active co-operation of all interested groups for effective action.

TABLE VII. PUBLIC NON-MOTOR VEHICLE DEATHS, BY OCCURRENCE, MINNESOTA, 1950

Cause	All Ages	Per Cent of Total	0-4 Years	5-14 Years	15-24 Years	25-44 Years	45-64 Years	65 & Over
Total	338	100.0	15	39	47	81	70	86
Falls	81	24.0	—	—	3	6	17	55
Drowning	80	23.7	11	24	15	9	13	8
Water transportation	52	15.4	—	2	11	23	14	2
Air transportation	36	10.6	—	4	4	23	4	1
Railroad, not with motor vehicle	26	7.7	1	1	—	5	8	11
Firearms	14	4.1	—	3	6	—	4	1
Other	49	14.5	3	5	8	15	10	8

It is of interest to note the small number of accidental deaths that occurred in mining, quarrying, construction, public utilities, and manufacturing. In spite of the hazardous nature of these occupations, they caused less than one-fourth of all occupational deaths. This record was undoubtedly due to the fact that industry has recognized the importance of accident prevention and has developed safety devices and safety education "on the job." The next step should be an extension of safety education into the homes of workers.

Public Accidents

Public accidents (non-motor vehicle) took 338 lives during 1950 (Table VII). The leading causes were falls, 24 per cent; drowning, 24 per cent; water transportation, 15 per cent; air transportation, 11 per cent; railroad transportation, 8 per cent; and firearms, 4 per cent. Of the fatalities from falls, more than two-thirds occurred in persons sixty-five years of age and older (68 per cent). Almost half of the public drowning occurred in children under fifteen years of age (44 per cent) and another one-fifth between fifteen and twenty-four years of age (19 per cent).

Obviously, an abnormal finding in the initial x-ray film cannot by itself constitute a clinical diagnosis (of tuberculosis). Nevertheless, it does serve to focus attention on those persons in need of further study. At the same time, such a photofluorographic finding imposes an obligation on the physician and the official health agency—an obligation to carry out full diagnostic study promptly in order to determine the clinical significance of the suspected abnormality. For, the abnormal findings discernible in an initial screening film can range all the way from minor deviations requiring no follow-up or care to some very serious conditions which demand immediate medical attention.— DAVID REISNER, M.D. and ARTHUR RIKLI, M.D., *Public Health Reports*, April 6, 1951.

THE USE OF WANGENSTEEN SUCTION IN SURGERY

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PROBABLY nothing has done more to facilitate the recovery of individuals subjected to abdominal procedures than the routine use of gastrointestinal suction in the immediate postoperative period. With elaboration of the idea of gastric suction by Wangensteen, a more rational concept of disturbed physiology following trauma to the intraabdominal viscera was possible. As a result of irritation of the peritoneum regardless of the cause, there is a functional inactivity of the gastrointestinal tract, which persists for varying periods of time according to the degree of trauma. In infections of the peritoneum, trauma to the peritoneum is prolonged and for this reason the associated adynamic ileus is of much more serious importance and significance than that which follows an operative procedure. Ever since introduction of gastric suction by Wangensteen, we have employed the Wangensteen tube routinely in all patients subjected to laparotomy except the simplest of procedures, such as an appendectomy. In this way we have been able to do away almost completely with postoperative nausea and vomiting, abdominal distention, and gastric dilatation. As a result of trauma and its associated ileus, the secretions which are normally poured into the upper part of the gastrointestinal tract, that is from the stomach, the duodenum, the liver and pancreas, and which normally are transported by peristalsis into the lower part of the intestinal tract from whence they are absorbed, remain in the upper part of the digestive tube and, if not removed, cause nausea and vomiting. By means of an indwelling catheter and suction, these secretions are removed and the stomach and upper intestinal tract are kept empty. In this way a persistent adynamic ileus is frequently prevented and resumption of normal gastrointestinal activity occurs early. The length of time gastric suction must be maintained varies according to the degree of trauma, but generally does not exceed twenty-four to seventy-two hours. By careful auscultation of the abdomen, one can determine the return of peristalsis, and as soon as peristaltic sounds become active, the tube can be removed safely.

Although there are some indications for the use of a double tube, these indications in our hands have become less frequent as time passes. In any case, however, in which decompression of the lower intestinal tract is desired and obtained by means of the double tube, it is imperative that gastric suction be maintained likewise, because otherwise accumulation of secretions in the upper gastrointestinal tract will occur and will be accompanied by nausea and vomiting. During the time that an indwelling gastric catheter is maintained and suction is applied, nothing is allowed by mouth, because otherwise large amounts of electrolytes will be washed out of the stomach by the gastric lavage. Obviously, the fluids and electrolytes removed by gastric suction must be replaced parenterally. If for any reason, there is not prompt resumption of gastrointestinal activity and prolonged gastric drainage is necessary, it is essential that potassium deficiency should be prevented by the administration of potassium intravenously. This is usually encountered only in those cases in which there is severe inflammatory disease producing prolonged irritation of the peritoneum and splanchnics.

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One check of the reliability of the 70 mm. film for the detection of tuberculosis by having two independent readings showed that the use of a second reader increased the number of suspected cases to be called back for verification by 46 per cent. The same study also revealed that those not reporting for examination were unrepresentative of the total population with respect to age and economic status. Based upon a sample who subsequently reported for examination, this group had a somewhat higher prevalence rate of tuberculosis.—HAROLD F. DORN, Ph.D., *American Journal of Public Health*, March, 1951.

PROCAINE AMIDE IN THE TREATMENT OF VENTRICULAR TACHYCARDIA

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PAROXYSMAL ventricular tachycardia may occur in persons without demonstrable organic heart disease.[2,8,11] Often it is encountered in arteriosclerotic and hypertensive heart disease and after overdigitalization.[13] A special type may be induced by emotion or exertion.[14] Most frequently the underlying cause is a recent myocardial infarction. It is justifiable to suspect every case of ventricular tachycardia to be caused by infarction until proven otherwise. The earliest sign of hyperirritability of the heart muscle is single or multifocal extrasystoles. Later on, series of ventricular extrasystoles ensue with normal rhythm between. The next step in the development is more prolonged attacks of ventricular tachycardia, lasting for days or weeks.

Experimentally, ventricular tachycardia may be induced by ligation of a coronary artery, by the administration of barium chloride, and by a combined stimulation of the vagus and accelerator nerves.[12] But the most famous experimentally produced ventricular tachycardia is the one induced by cyclopropane-epinephrine and chloroform-epinephrine anesthesia.[1,4,7] Benzol has also the ability to increase ventricular irritability. Mechanical irritation of the heart, as in heart catheterization, may lead to ventricular premature beats and ventricular tachycardia. Electrical shock may bring about ventricular fibrillation.[5]

The occurrence of cardiac arrhythmias has been reported in myocardial infarction in 9 to 27 per cent of the cases, with an average of 18 per cent, and ventricular tachycardia may develop in approximately 1 per cent of the cases.[14] It is always a serious condition, indicating a severe degree of irritability of the heart muscle and frequently terminating fatally in ventricular fibrillation. In Williams and Ellis' series consisting of thirty-six patients, eight died during the attack and thirteen more shortly after attacks. If the attack lasts over a long period of time, danger of heart failure is always present, because of the insufficient filling of the heart during diastole.

For many years procaine has been known for its ability to prevent and control cardiac arrhythmias in anesthetized patients.[6] Its usefulness, however, is limited because of its rapid destruction and elimination from the body as procaine esterase and by its hypotensive action. In unanesthetized patients, it acts as a central nervous stimulant with tendency to convulsions. Marks et al[9] have studied the effect of various chemical compounds belonging to the diethylaminoethanol-group on ventricular tachycardias experimentally produced in dogs by cyclopropane-epinephrine anesthesia. The most promising one was procaine amide (Pronestyl Squibb). It may be given orally as well as intravenously, is rapidly and completely absorbed from the intestines and is not hydrolyzed by the plasma enzymes. The plasma levels decline 10 to 15 per cent per hour, and about 60 per cent is excreted unchanged in urine. The toxicity of procaine amide seems to be small. Doses of from 3 to 6 grams per day have been given orally from two days to three months without toxic effects. It has been found to cause a drop in blood pressure in many cases when administered intravenously. This route of administration should therefore be used with great caution, and the drug should be injected very slowly.

Because of the good results that have been reported in the literature lately, it was decided to give procaine amide a trial on a patient with ventricular tachycardia who came under our treatment recently:

The patient, a white man, sixty-seven years old, was treated in Asbury Hospital from February 2 to March 11, 1951, for myocardial infarction.

A known hypertensive for years, he walked one mile in 20 below zero weather on February 1 in the afternoon without having any complaints at that time. In the evening, however, he developed pain across the chest, radiating to the left shoulder and left arm, to the neck and to the left side of the lower jaw, where the pain was felt like a toothache. He broke out into a sweat. Pain grew worse and he was unable to find rest in any position. A doctor was called. At that time his color was good, skin warm and dry, temperature 98.6, pulse 72 and regular, blood pressure 200/100. He was given a sedative after which he became nauseated and vomited and felt somewhat better.

The next day he had a chill, and his only complaint was a dull ache over the left lateral chest. Early in the

Case report through courtesy of J. J. Kaplan, M.D.

morning of February 3 he developed intermittent cramping pain in the abdomen which lasted through the night. He was again seen by his doctor who found his temperature 100.2, pulse 84 and regular, blood pressure 140/90, color good. He had crepitant râles over both lung bases. Heart sounds were of good quality with a systolic murmur heard over the mitral area; normal rhythm. No edema was present. There was an umbilical hernia and bilateral inguinal hernias.

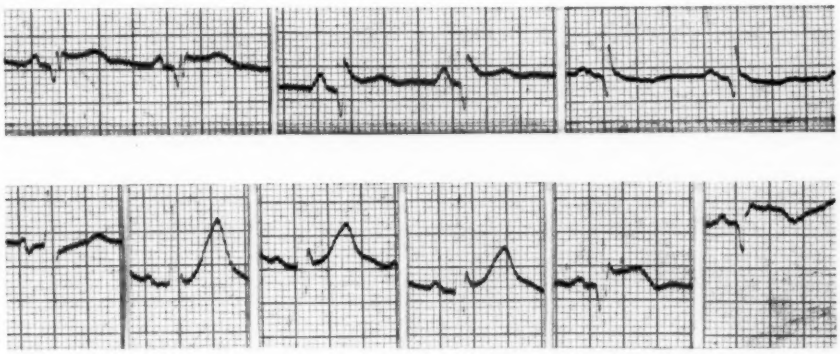

Fig. 1. Electrocardiogram taken the day of admission, showing evidence of anterolateral infarction. First row, limb leads I, II, and III. Second row, leads V 1 to V 6.

On the day of his arrival at the hospital he complained of severe chest pain and was given 100 mg. of Demerol. He obtained relief and the pain did not recur. Examination now revealed a blood pressure of 120/60, but otherwise no change from the description above. His sedimentation rate was 60 mm. in one hour, white blood count 25,950 with 91 per cent polymorphonuclears and 9 per cent lymphocytes. Urine was normal. Electrocardiogram was consistent with an acute anterolateral infarction (Fig. 1). X-ray of the chest showed a marked left ventricular cardiac enlargement and slight pulmonary congestion.

He was given the usual treatment with bed rest, anticoagulants, antispasmodics and in addition penicillin. On February 5 a precordial friction rub was discovered and it was present for about ten days. On February 6 the friction rub was heard over the entire precordium and the patient had a pectoriloquy over the left lung posteriorly, thought to be caused by a pericardial effusion with compression of the lower left lung lobe. Another roentgenogram showed some haziness over the right lower lung field, suggestive of possible infarction.

He raised some mucopurulent sputum the first two to three days in the hospital, but no bloody sputum was observed. His white blood count fell to 20,000 and the sedimentation rate increased to 73 mm. on February 5. Five days later the white blood count was 16,200 and sedimentation rate 108 mm. His temperature was elevated to 100.4 for the first nine days he spent in the hospital; later it was normal. His blood pressure fell to 100/70 the second day after admission and has since been fluctuating between 92 and 120 systolic and 50 and 90 diastolic. His pulse ran between 70 and 90 for the first four days.

On the morning of February 8 a pulse rate of 40 to 44 was discovered. His heart rate, however, was between 80 and 90. His blood pressure was 114/70. It was evident that every second beat was an extrasystole, but the premature beat occurred so early in the diastole as to provoke no pulse wave. An electrocardiogram confirmed this by showing electrical bigemini throughout (Fig. 2). He was given quinidine sulfate gr. III q.i.d. from now on. He was also given Digitoxin 0.2 mg. for three doses only, this same day.

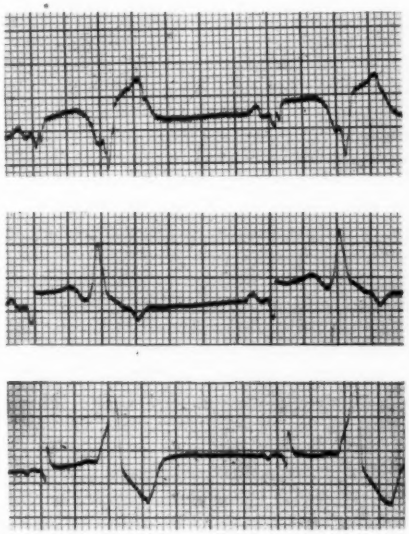

Fig. 2. Electrocardiogram five days after admission, showing electrical bigemini. Limb leads I, II, and III.

For the next two days, he showed intermittent bigemini. At 7 a.m. on February 11, it was difficult to obtain his pulse and the heart rate was about 200 per minute. Blood pressure was 88/68. An electrocardiogram showed ventricular tachycardia. Oxygen was started. At 9:30 a.m. while he still had ventricular tachycardia Pronestyl hydrochloride was given intra-

venously slowly. The injection was stopped after 500 mg. had been injected, and at the same time a change to sinus rhythm had occurred. His blood pressure was between 80 and 90 systolic while the injection was given, but increased to 106 systolic at the moment a sinus rhythm was restored. The pulse was practically imperceptible while the tachycardia lasted but became strong, regular and about 72 after reversion to sinus rhythm. He was given orally continuation doses of Pronestyl hydrochloride 500 mg. at 9:45 a.m. and 250 mg. at 2:30 p.m., 6:30 p.m. and 10 p.m. At 1:10 p.m. the special nurse again noted bigemini. Another electrocardiogram confirmed this. At 9:30 p.m. ventricular tachycardia again was present. Pronestyl hydrochloride was given intravenously for the second time, and after injection of 500 mg. sinus rhythm was restored. An electrocardiogram was taken continuously during the injection and showed the sudden change in rhythm (Fig. 3). He was given a larger maintenance dose consisting of 500 mg. Pronestyl hydrochloride orally every four hours for three days. The dose was then reduced to 250 mg. every four hours for three days and after that 250 mg. four times daily for five days.

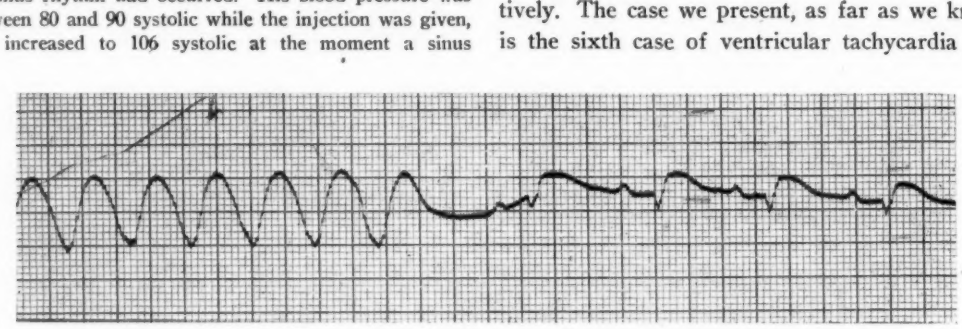

Fig. 3. Electrocardiogram eight days after admission showing reversion of ventricular tachycardia to sinus rhythm as procaine amide was injected intravenously. Limb lead II.

Medication was stopped on February 22. He received no more quinidine after he developed ventricular tachycardia. No more disturbances of the heart rhythm could be detected by us, the pulse rate being between 60 and 80 and regular. He had some urinary difficulty and had to have an indwelling catheter for five days. He developed urinary infection and was treated with sulfadiazine, but his recovery was otherwise completely uneventful, and he left the hospital steadly improving, on March 11.

There are few reports in the literature so far about procaine amide treatment of patients with cardiac arrhythmias. L. C. Mark et al. were able to suppress premature ventricular beats in patients for varying lengths of time by oral or intravenous medication. They were able to terminate prolonged attacks of ventricular tachycardia in three of four patients. Two of these had previously received quinidine to toxicity without effect. J. M. Kinsman et al. succeeded in reverting the rhythm to normal in two patients with paroxysmal ventricular tachycardia promptly after administering 400 and 550 mg. procaine, respectively. The case we present, as far as we know, is the sixth case of ventricular tachycardia that has been successfully treated with procaine amide. Our patient has also been treated with quinidine, which in smaller doses was unable to prevent the occurrence of the tachycardia. We did not try larger doses of quinidine and are therefore unable to judge whether this therapy would have succeeded or not. J. M. Kinsman et al. had also good experience in the treatment of ten of eleven patients with premature ventricular contractions by means of intravenous administration of procaine amide. The drug does not seem to have the same over-all success in the treatment of disturbances of auricular rhythm. Kinsman found no benefit in auricular flutter and fibrillation and auricular tachycardia. Of four patients with auricular premature beats, however, the abnormal rhythm was abolished in three cases. The same author noted the occurrence of transitory bundle branch block after procaine amide administration, which indicates a depressant action upon the specific conduction tissue in the heart, while the above-mentioned effect upon ventricular premature beats and ventricular tachycardia is due to a depression of the irritability of the contractile tissue of the heart.

As soon as this patient showed bigemini, we knew that hyperirritability was present and that he could be in danger. Quinidine was given to lessen this danger. We are fully aware of the ability of digitalis to increase the irritability of the heart muscle especially when it is given in toxic doses. On the other hand, we have been able to demonstrate repeatedly disappearance of arrhythmias in patients with enlarged heart and heart failure when given digitalis. In Williams and

Ellis' thirty-six cases of ventricular tachycardia, seventeen had received digitalis medication, and eight showed signs of digitalis toxicity. In our case, it was used in only half digitalization dose. We are unable to tell what role digitoxin played in the development of the ventricular tachycardia in this case, but we agree that it should not be given unless strong indications exist for its use. We did not try other drugs which have been used in this arrhythmia. It is our opinion that the recurrence of bigemini and later ventricular tachycardia in this patient was caused by too small maintenance doses of procaine amide. We are justified in this opinion by the restoration of sinus rhythm during the second injection and no later occurrence of any arrhythmias when the maintenance doses were increased. These doses will undoubtedly vary from patient to patient, depending upon the degree of hyperirritability of the heart muscle. It is the attending physician's job to find out the maintenance dose. We could not detect any untoward reactions to the drug. There was no lowering of the blood pressure which may be seen when larger doses are given intravenously. On the contrary, we observed a rise in blood pressure on both occasions, believed to be caused by better filling of the heart during diastole when the rate and rhythm reverted to normal.

Summary

A case of myocardial infarction is described in which bigemini developed on the seventh day after onset of the disease. Under quinidine treatment intermittent bigemini was observed.

On the tenth day after onset of the disease ventricular tachycardia developed. Sinus rhythm was restored with 500 mg. procaine amide intravenously. The oral maintenance doses of this drug were apparently too small, because bigemini and ventricular tachycardia recurred later on the same day.

Again restoration of normal rhythm was obtained with intravenous injection of 500 mg. of procaine amide. Larger maintenance doses were given orally with gradual tapering off, and no further arrhythmias were observed. The patient made a good recovery.

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Throughout the years, there has been a definite line of separation between the functions of the hospital and those of the health department. Health departments accepted the responsibility for the control of communicable disease within the community while hospitals confined themselves to the diagnosis and treatment of disease of patients within the institution. In recent years, there has been an awakening to the fact that these traditional functions must be broadened and integrated if the maximum benefit to the health of the nation is to be achieved. In the field of tuberculosis control, this coordination of activities is essential. The hospitalization of the tuberculous patient is but one phase of the total care. The hospitalization cannot be carried out most effectively without relationship to what precedes and follows in the life of the patient.—*Public Health Nursing*, ELVA M. LEWIS, R.N., August, 1950.

THE FIRST CONTRIBUTION TO A MEDICAL JOURNAL FROM MINNESOTA

An Epidemic of Scarlet Fever at Fort Snelling in 1847-1848

JAMES ECKMAN, Ph.D.

Rochester, Minnesota

> In the *Youth* of a *State Arms* do flourish; in the *middle Age* of a *State Learning*, and then both of them together for a time: in the *declining* Age of a *State, Mechanical Arts* and *Merchandise*.
>
> —FRANCIS BACON[3]

ONE MARCH day in 1848, in the wonderful year of Manifest Destiny of what has been called the era of "the rise of the common man,"[13] an Army surgeon at Fort Snelling in the Territory of Wisconsin sat writing at a report that had occupied his attention for some time. What was to be the city of Saint Paul was in that year "a mere collection of huts with Indians and birch-bark canoes promiscuously plenty,"[38] with scarcely a sawed frame building in the place. The first regular line of steamboats between Saint Paul and settlements down the Mississippi River had been in operation only about a year; the post office in Saint Paul was hardly two years old.[15] In the entire area which a year later was to become the Territory of Minnesota there may have been some 4,000 white or mixed-blood settlers, none of them legally established, for despite a mounting pressure occasioned by the lure of free land, the region was not yet open to settlement by the white man.[15] In Saint Paul there were surely no more than three physicians;[1] in Saint Anthony none at all.[22]

Far to the east, on that March day, James Knox Polk of Tennessee, eleventh President of the United States, was sitting for his portrait in the White House.[8] He was somewhat relieved to reflect that the treaty ending the late war with Mexico had been ratified by the Senate,* but he was still fretting over the hordes of predaceous office-seekers constantly plaguing him. On that March day, also, General Winfield Scott, the choleric Whig conqueror of Mexico, was departing from Mexico City to return to the United States, recalled by order of the President who had not been able to find a Democratic general to conduct the war for him.[11] Robert E. Lee, a captain of engineers, at the time was engaged in a methodical cartographic survey of the hills west of the Mexican capital;[16] Ulysses S. Grant, a first lieutenant of the Seventh Infantry, was whiling away his time in Mexico by climbing Popocatepetl and visiting the ancient tombs near the beautiful little city of Cuernavaca.[20] General Zachary Taylor, hero of Buena Vista and the next President, was in Louisiana, fencing rather elegantly by correspondence with Henry Clay on the subject of whom the Whigs would nominate for the Presidency in November.[7] In Washington Abe Lincoln of Illinois was sitting as the only Whig member of Congress from his state.[44] The godlike Daniel Webster, senator from Massachusetts, afflicted with "some lingering rheumatism" and catarrh, was preparing to leave Washington for Boston, alarmed at the serious illness of a daughter.[65] In New York City the aged John Jacob Astor lay on his deathbed;[36] only a month previously John Quincy Adams had died in the Speaker's Chamber of the Capitol in Washington.[43] On the opposite shores of the continent men were contesting frenetically for the yellow gold that had been discovered

From the Section of Publications, the Mayo Clinic.

The author wishes to acknowledge his gratitude to Dr. Philip M. Hamer and Miss Elizabeth B. Drewry, of the National Archives, Washington, D. C., for making available to him the correspondence between Dr. George F. Turner and the Office of the Surgeon General, U. S. Army; Miss Lucile M. Kane, curator of manuscripts, the Minnesota Historical Society, for permitting him to examine the Franklin Steele Papers and the H. H. Sibley Papers owned by the society; to Dr. Reginald Fitz, assistant dean of the Harvard Medical School, for making available certain manuscript sources concerning Dr. Turner; and to the Boston Public Library and the Library of Congress, Washington, D. C., for allowing him to examine newspapers and other material of the period embraced by this paper.

*The Treaty of Guadalupe Hidalgo was submitted to the United States Senate on February 23, 1848; ratified by the United States on March 16 and by Mexico on May 30, and was proclaimed to be in effect on July 4, 1848. It is interesting to notice that although this treaty was ratified by the United States in March and by Mexico in May, 1848, William T. Sherman, at the time a first lieutenant of the 3rd Artillery stationed at Monterey, California, wrote that he did not receive news of the ratification until September, 1848. *See:* Miller, Hunter, ed.: Treaties and Other International Acts of the United States of America. Washington, Government Printing Office, 1937, vol. 5, pp. 207-428. *See also:* Memoirs of Gen. W. T. Sherman, Written by Himself. New York, Charles L. Webster & Co., 1891, vol. 1, p. 85.

in California some two months earlier; but in the placid Mississippi Valley no one had yet heard the electrifying news.** In London the celebrated historian, George Bancroft, Polk's minister to England, was recording with elation for Secretary of State Buchanan the news of the street fighting in Paris which toppled the bourgeois government of Louis-Philippe and presaged other uprisings in Austria, Germany, Italy, Denmark and Poland in that memorable year of the revolutions of 1848.[39]

But at the remote territorial military post of Fort Snelling there were neither momentous events nor formidable personages to relieve the monotony of the March day. When the Army surgeon had finished his manuscript, he sent it, perhaps by river boat, to the *New-York Journal of Medicine and the Collateral Sciences.*[47] Armstrong[1] regarded the paper as perhaps "the first contribution to a medical periodical from Minnesota." The text follows in full.

ART. III.—*An Account of a severe Form of Epidemic Miliary Fever, which appeared at Fort Snelling, Upper Mississippi, in the Winter of* 1847-48.

By GEORGE F. TURNER, M.D., Surgeon, U. S. Army.
Fort Snelling, March 22d, 1848.

To the Editor of the
New-York Journal of Medicine, &c.

Dear Sir,—I have been induced to send you this imperfect sketch, from the belief that the severe form in which this epidemic has appeared is peculiar, and because very high authorities have denied the existence of miliary fever as a primary disease. I do not know that it will be of any interest to your readers.

During the past winter there has prevailed here, and in this vicinity, an epidemic miliaria of serious character, confined almost exclusively to children, which, so far as I know, is in many respects peculiar.

The first two cases occurred almost simultaneously, about the 20th December, 1847, in children of the same family, and proved rapidly fatal, and since that time the disease has moved slowly but steadily on, attacking family after family, till there now remain but few subjects for its visitation.

In its access it resembles the eruptive fevers generally; the leading symptoms being, in a majority of cases, vertigo, drowsiness, rigors alternating with flushings, pains in the back and lower extremities, great lassitude, coldness of the feet and hands, followed generally in the course of twenty-four hours, by soreness of the throat, the inflammation usually beginning in the tonsils, which are early dotted by ulcerated points, the fauces soon becoming inflamed, and subsequently the mucous membrane of the nose. The tongue at this stage usually presents a peculiar appearance, as if rubbed with chalk; the papillae showing above the white ground of a dark red hue, and the breath is now intolerably offensive. The febrile action, which is highly inflammatory, being now fully developed, the skin is hot and red, accompanied by a sense of pricking and itching which is sometimes excessive, and delirium is apt to be present, and in favorable cases the eruption (attended by a general roughness of the surface, constituting cutis anserina), begins to appear within forty-eight hours from the first indisposition. The eruption shows itself first on the loins, and extends gradually over the back and abdomen, and often over the entire surface; the face being the last to show the rash. It is distinctly vesicular, rendering the skin rough and of a deep red; disappearing on one part and appearing on another; the desquamation, in branny scales, commencing in favorable cases about the third day from its first appearance; though in many cases the eruption has re-appeared from time to time, not in the vesicular form but merely showing beneath the cuticle, for three, four, and five weeks after the subsidence of the original disease. In one case a second eruption occurred three weeks after the disappearance of the first, closely resembling rubeola; the patches being interspersed with the weals of urticaria, and this was fatal, from the supervention of peritonitis on the sudden retrocession of the eruption.

An excessive eruption has not been as favorable as when less extensive, leaving one part to appear on another. The former cases have been attended by an increased degree of inflammation of the fauces and mucous membrane of the nose, and have been followed by more and severer sequelae.

The most severe cases have been those where the eruption from the first has been barely visible, and where gastric irritability has been great.

With a few exceptions, bronchial symptoms have been slight, or altogether wanting.

In the most favorable cases convalescence has commenced about the seventh or eighth day, though few cases have progressed to perfect recovery without relapses from sequelae more painful and serious than the original disease. In fact, this disease (except where alarming symptoms have early appeared), has not assumed its most formidable character until after the eruptive stage has terminated. Convalescence has been tedious and uncertain; and enlargements of the parotid and cervical glands, otitis, with abscess within the ear, muscular pains resembling rheumatism, and anasarca, have formed the usual sequel. Of the forty-two cases which have come under my care, thirty-seven have been children under twelve years old (and this number embraces nearly all the children in this immediate vicinity), four were adult females, and one male. Six deaths have occurred thus far. One from laryngitis, one from meningitis, two from bronchitis, all rapidly fatal (the eruption having never been fully developed, but only slightly perceptible, and that in a very limited extent), and two from peritonitis, relapsed cases, after four or five weeks from the subsidence of the original eruption. Many cases which have recovered have been of

**The Mississippi Valley first received news of the discovery of gold in California in August and September of 1848. The exodus of gold seekers from this region to the Pacific Coast apparently reached greatest proportions between February and May, 1849. See reference 4.

a most serious character, and altogether the disease has been formidable. As to its mode of propagation, I cannot compare it with any epidemic I have seen. As a general rule, where it has entered a family, none of the children have escaped, but have all suffered within from three to six or seven days; though there have been a few striking exceptions. In one family, two children had the disease, and after an interval of seven weeks another took it, and then a fourth after the lapse of five weeks, and a fifth a few days subsequently; the remaining three having thus far escaped.

In this garrison, of eleven children occupying adjoining quarters on one side of the parade, ten were attacked, and not till six weeks after the last case, did the children on the opposite side begin to suffer.

The disease is probably, under favoring circumstances, slightly contagious. I have seen, in previous years, isolated cases, and its prevalance this season seems to depend on some inappreciable atmospheric peculiarity.

It has prevailed epidemically among the Sioux Indians of this vicinity while on their winter hunt, attacking indiscriminately all ages and sexes; but with them, from causes probably connected with their mode of life, it has been comparatively mild. In the memory of the oldest Indians, this epidemic has occurred amongst them twice before, and is peculiar to the cold months in this climate.

The treatment of this disease has been dictated by general principles. Emetics of ipecac, when not contraindicated by gastric irritation, have proved of decided benefit, when given at the very onset, and subsequently mild cathartics through the whole course of the disease; though the persevering use of tepid sponging has been of more essential service than any other single remedy; allaying excessive heat of the skin, and general vascular excitement, obviating delirium and restlessness, and promoting sleep; thereby diminishing materially the risk of local congestions and inflammations; which have constituted the chief danger in recent cases.

Rooms highly heated by stoves, with imperfect ventilation, and too much bed covering, have proved in the highest degree pernicious during the eruptive stage; aggravating in a marked degree every serious tendency.

During the tedious and lingering convalescence, tonic, and alterant treatment has been indicated.*

Who was Dr. George F. Turner, surgeon of the United States Army? Assiduous biographers, after months or possibly years of exhaustive and exhausting toil, too often become infatuated with the subject of their researches, their devotion impelling them to fashion him into a stature which he plainly did not possess. Let it be said at the outset that George F. Turner was not an important figure in American history or in American medicine. Like those other Army officers now all but forgotten—Sully, Kearny, Harney, Crook—Turner was remarkable only within his own periphery and his periphery was extremely limited.

Such eminence as should be accorded him derives chiefly from the fact that in a ruthless, riotous and at times almost overwhelming wilderness scene he was a Harvard-trained physician on a savage frontier where murder, strife and inebriety more often than not demanded his best efforts. He was also, incidentally, the surgeon who at Fort Snelling succeeded Dr. John Emerson, the physician whose name today is quite obscured by that of his black bondsman, Dred Scott.

The assistant dean[14] of the Harvard Medical School discovered and very kindly furnished a manuscript memoir of Dr. Turner written in 1855 by Lt. Col. William Gouverneur Morris.[35] Morris wrote that Turner was born in Boston on April 22, 1807, although records of the Harvard Medical School show that when Turner matriculated there on November 15, 1826, he gave his residence as Richmond, Virginia.[14] Army records indicate that Turner was born in Boston.[68]

His preceptors were two of the most distinguished men in American medicine: Dr. Walter Channing (1786-1876), professor of obstetrics and medical jurisprudence at Harvard from 1815 to 1854, and one of the first men to advocate the use of ether to alleviate the pain of childbirth; and Dr. James Jackson (1777-1867), professor of clinical medicine at Harvard, acting physician to the Massachusetts General Hospital from 1821 to 1837, and one of the group which founded what, in 1828, became the *Boston Medical and Surgical Journal*.[42]

Turner as a student at Harvard was characterized by someone as "Gentlemanly, kind hearted, noble hearted, full of fun & waggery, honorable, dyspeptic, respectable but not distinguished in medical . . ."*[35] On January 26, 1830, Turner was examined by the faculty of the Harvard

*The late Dr. C. Anderson Aldrich, director of the Rochester Child Health Institute, Rochester, Minnesota, and a member of the Section of Pediatrics of the Mayo Clinic, examined this paper and commented on April 15, 1948: ". . . after reading it over carefully I should say that this corresponds very closely with what we would now call scarlet fever, although some of the characteristics are not typical. As to the treatment with ipecac and cathartics I certainly think they would not be considered good treatment today. The sponges of course are all right. It is strange that the Doctor says nothing of nephritis as a complication which one would expect to see popping up in as many severe cases as he saw. While it appears dangerous to make a diagnosis one hundred years following an epidemic I don't think many doctors would doubt the diagnosis of scarlet fever in this case."

*Illegible in the manuscript by Lt. Col. Morris.

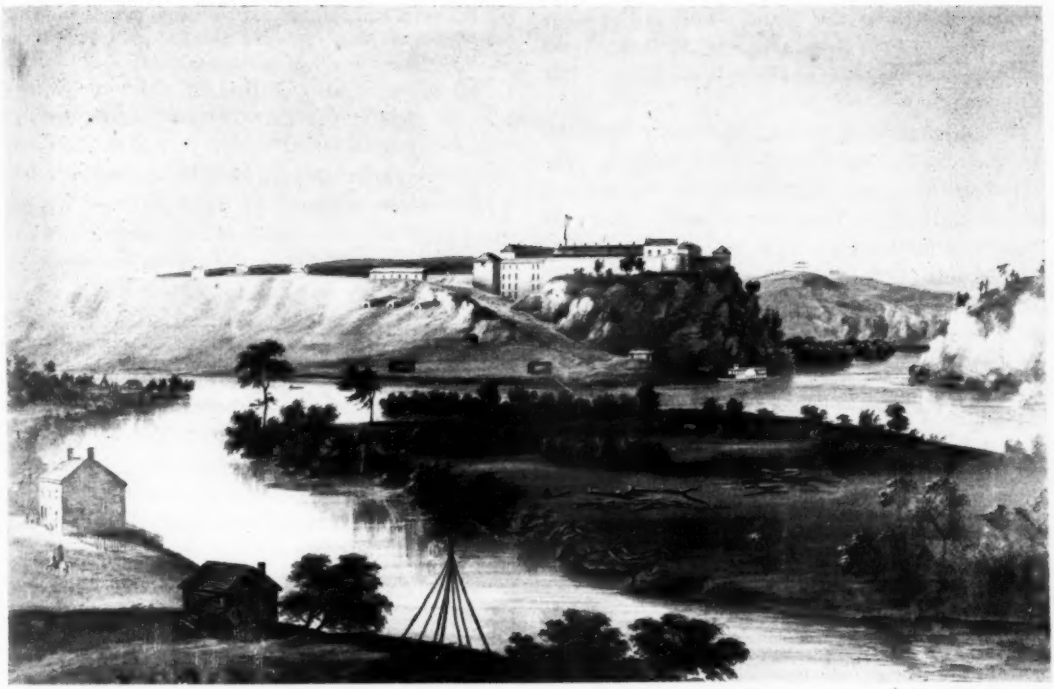

Fig. 1. Painting of Fort Snelling believed to have been done in 1844 by John Caspar Wild (1804-1846), a Swiss painter and lithographer who had settled in Saint Louis. Dr. Turner had been at Fort Snelling for four years when this view was painted. Reproduced by permission of the Minnesota Historical Society, which owns the painting. *See:* McDermott, J. F.: J. C. Wild and Fort Snelling. Minnesota History. 32:12-14 (spring) 1951.

Medical School and approved for public examination. The subject he had chosen was "Hemorrhoids," and on February 3, 1830, he successfully defended his dissertation on this subject at public examination and was recommended for the degree of doctor of medicine.†[14] It is said[35] that after receiving his doctorate he practiced medicine for awhile in Indiana; if so, no record of it is at hand. He was commissioned an assistant surgeon in the United States Army on July 23, 1833, with the grade of captain.[68]

His pay could not be regarded as exceptional. An assistant surgeon of less than five years' service in 1834 received the pay and emoluments of a first lieutenant of the line, meaning $30 a month as salary, two rations a day and forage. One ration consisted of 1¼ pounds of beef or ¾ pound of pork, 18 ounces of bread or flour, and 1 gill of rum, whisky or brandy. For every 100 rations he was entitled to 2 quarts of salt, 4 quarts of vinegar, 4 pounds of soap and 1½ pounds of candles.[2]

In November, 1833, he was sent to Fort Mackinac, Michigan, where in 1834 he married the eldest daughter of Mr. Robert Stuart, of Detroit.[35] In December, 1835, and January, 1836, he was stationed at Castle Pinckney, South Carolina, overlooking the city of Charleston.[9] For a year, February, 1836, to February, 1837, Dr. Turner was stationed in Florida, to which he returned in September of 1837. He served in Florida through June, 1838, and in July was ordered to Governor's Island, New York. In September, 1838, Dr. Turner was sent once again to Florida, where he remained until July, 1839. Surgeon Robert C. Wood (1800-1869), who had been stationed at Fort Snelling in 1829 and was assistant surgeon general of the Army from 1862 to 1865, was in Florida with Turner. He ranked thirteenth among

†Another Harvard-trained man who came to Fort Snelling in an early day was John Marsh (1778-1856), of Salem, Massachusetts. Marsh received the degree of bachelor of arts from Harvard College in 1823, three years before Turner enrolled in the Harvard Medical School. Marsh was at Fort Snelling from 1823 to 1825, where he was post schoolmaster and for a time acting Sioux Indian agent. He studied medicine under Dr. Edward Purcell, post surgeon at Fort Snelling, and never obtained the degree of doctor of medicine. *See:* Lyman, G. D.: John Marsh, Pioneer. The Life Story of a Trail-blazer on Six Frontiers. New York, Charles Scribner's Sons, 1930. 394 pp.

medical officers of the Army. Turner doubtless knew him well.*[40] From October, 1839, to April, 1840, Turner was stationed at West Point, New York.[9]

On March 7, 1840, Dr. Turner was notified by Dr. Thomas Lawson (1789-1861), surgeon general of the Army, that he would shortly be ordered to Fort Snelling, at that time in the Territory of Iowa.[27] Whether or not a prospective tour of duty in such a far-distant post played a part in his advancement is not known, but on January 1 of that year he had been promoted to the status of surgeon, with the grade of major.[68] On April 16, however, Turner was still at West Point, complaining to Lawson that his orders had not yet arrived, and that he wished to take the steamboat *Pennsylvania* at Pittsburgh on May 5, for Fort Snelling.[53] It was this vessel which had brought Brigadier General John E. Wool (1784-1869), inspector-general of the Army, to Fort Snelling in the preceding summer on a tour of inspection.[37]

By April 24 Dr. Turner was in Pittsburgh.[54] He relieved Surgeon John Emerson at Fort Snelling on May 29, 1840.[29] Oddly enough, Turner seems to have been unable to remember when he reached the fort: on one occasion he wrote that he "received of Dr. Emerson when relieved by me on 29th June 1840 $26.50,"[55] but on another he reported that he had received stores from Dr. Emerson in May, 1839,[56] which obviously was impossible. The date given by Surgeon General Lawson[29] (May 29, 1840) undoubtedly is the correct one.

It is possible that as the steamboat *Pennsylvania* drew near the confluence of the Minnesota River with the Mississippi, the Army surgeon may have experienced an emotion similar to that of a boy[5] who had first beheld the fort (Fig. 1) from the deck of another steamboat eight years previously:

> A sight never to be forgotten was when on turning a point in the river there suddenly appeared, a mile or so before us, the imposing and beautiful white walls of Fort Snelling, holding, as though by main force, its position on a high, precipitous bluff, and proudly floating the stripes and stars. It was a fortified oasis of civilization in a lovely desert of barbarism.

But it might also be that the "desert of barbarism" was a prospect dismal enough to the young physician from Boston. For a decade prior to his arrival at the fort in 1840 the irrepressible tide of those attracted to the Northwest by the great magnet of free land had been beating against the barrier constituted by the military reservation of Fort Snelling and the forbidden Indian lands beyond. Every summer, between 1830 and 1850, the number of new farms wrested from the soil in the great plains north of the Ohio and the Missouri rivers averaged about 20,000.[13a]

Only a fortnight before Dr. Turner arrived, the troops at Fort Snelling had been called out to dispossess whisky-selling white squatters from the military reservation.[37] Some of them were so disreputable that Turner's predecessor, Surgeon Emerson, had complained to Surgeon General Lawson of "the most beastly scenes of intoxication among the soldiers of this garrison and the Indians in this vicinity"; a sergeant in fact was so fond of the *aguardiente* that he had been willing to pay $80 for a gallon of it.[66]

Zachary Taylor, a decade earlier, had found the fort "a most miserable & uninteresting county" in an "out of the way part of the world."[23] Captain Frederick Marryat, author of *Midshipman Easy*, who had visited the fort two years before Dr. Turner reached it, liked it so much that he could have remained there a year with pleasure; the Indians, he thought, "must be acknowledged the most perfect gentlemen in America. . . ."[34]

But Turner himself seems to have had no very high regard for the place. To General H. H. Sibley in Washington he wrote:[64]

> We of the garrison are as usual at this season rather dull & unprofitable—small parties for Tea are a good deal the fashion, & tattle is used as formerly. Indian ball plays* are coming in season. One comes off today

*"The service in Florida, to most of the medical officers employed there," Surgeon General Lawson wrote, "has been indeed not only irksome, but exceedingly laborious and hazardous, many of them having from the very dispersed state of the troops, to give their attendance to two, three or more posts or stands; frequently passing from one station to another without an escort and occasionally under the fire of the enemy [the Seminole Indians]." *See:* Ashburn, P. M.: A History of the Medical Department of the United States Army. Boston, Houghton, Mifflin Co., 1929, p. 54.

*Captain Seth Eastman (1808-1875), one of the finest painters of Indian scenes America ever produced, achieved what some consider his most successful painting in his "Squaws Playing Ball on the Prairie," now owned by the Peabody Museum of Harvard University. The painting depicts an Indian ballgame in which all the players are women; the locale probably is a plain before Fort Crawford. Eastman was at Fort Snelling from 1841 to 1846, and then, after a brief interruption, until 1848. His tour of duty at that place thus coincides almost exactly with that of Dr. Turner, who arrived in 1840 and left in 1848, but no mention of Captain Eastman is made in any of Dr. Turner's papers that have come to hand.

in which stacks of property are to be invested. . . . What is the use in writing to you as I cannot find enough wherewith to fill two pages.

Dr. Turner was scarcely established at the fort before he was in trouble with the government. His name, it seems, had appeared on two different sets of pay accounts for April, 1840, and Surgeon General Lawson was properly and even punctiliously inquisitive[28]

Dr. Turner's explanation is not known. But throughout their correspondence, over many years, Surgeon General Lawson constantly upbraids Dr. Turner for minor derelictions, most of them involving only deviations from tedious Army routine.*

Lawson apparently scented a serious discrepancy when he discovered that Dr. Turner had fitted a citizen near the fort with a truss, charging him $3.00 for it. Turner replied that the man could not have obtained one except by going down the Mississippi to Galena for it, and since the man was suffering severe pain from a recent inguinal hernia, he thought circumstances warranted what he did.[58] The sum of $8.00, credited by Dr. Turner to the hospital fund, puzzled Surgeon General Lawson; Turner explained that it was paid to him by a citizen admitted to the fort "in a most deplorable condition from twenty miles distant," there being no other place at which he could be treated.[58] "As you were prompted, in both cases by motives of humanity which you could not with propriety disregard," the surgeon general sententiously wrote to Turner, "your conduct meets with my approbation."[31]

On another occasion in 1841 Acting Surgeon General Henry L. Heiskell reproved Dr. Turner because one set of routine papers from Fort Snelling was fifteen days late, and another was thirty-nine days late. "It is presumed," Heiskell wrote, in the chill, lifeless prose of desiccated officialdom, "you have some satisfactory reason for your dilatoriness; but as you have not thought proper to offer any, you are required immediately to explain the cause of the extraordinary delay in forwarding your Returns and Reports."[24] The "extraordinary delay" proved easy enough to explain. Dr. Turner's hospital steward had been ordered to Florida with the Fifth Infantry, and Turner had to assume his duties in addition to his own. He himself had been afflicted with inflamed hemorrhoids which would not allow him to either sit or stand; they in turn engendered a state which was designated as a neuralgic condition of the rectum; this confined him to bed for two months. During this period he had to arise each morning to serve for a short time as physician to the sick of the command as best he could; ill as he was, there was no physician to treat him.[48] In addition, he later reminded the surgeon general, "As the mails leave Fort Snelling but twice in the month, it may not always be possible to transmit my Returns, Reports &c. to your office within the period prescribed by regulations."[49]

On still another occasion Dr. Turner was audacious enough to send in an annual requisition written on a blank sheet torn from a bound book. This was too heinous a breach to pass unnoticed; the surgeon general remonstrated with the frontier Army physician. Turner's reply[56] was restrained:

> I have the honor to acknowledge the receipt of your communication of 29th Oct. 1842 containing in a postscript a rebuke in relation to one of my Annual Requisitions, which was made out on a Sheet from a bound book. The book in question when I removed a Sheet from it had been in use between six and seven years, and was full every page of it except two Blank Reports of Sick (of the old form), and three Blank Requisitions. The book when I received it of Dr. Emerson in May 1839* was in a most dilapidated state—a large portion of its leaves being partly or entirely separated from the binding, and moreover it had been for some time laid aside on the supposition that it was unfit for further use. Under these circumstances I could perceive no objection to applying a sheet of it to a useful purpose.

But Turner's troubles continued. He installed a barometer "in a large Strong wooden box having a latticed door—This box is securely fastened to the outer wall of the Hospital [Fig. 2] under

*The meticulous attention which Surgeon General Lawson devoted to Dr. Turner's relatively inconsequential faults should have surprised no one who was aware of the surgeon general's crochety temperament. Lawson served as surgeon general for twenty-five years, which means that for most of that time he was seated behind a desk in Washington. "General Lawson was rather of an administrative than a literary turn of mind," Pilcher wrote, "although he wielded a trenchant pen when occasion seemed to demand it. . . . He was a pronounced adherent of 'the code' and thought that medical officers should be as ready as those of the line thus to repel and resent insult, believing that much of his success as commander of troops would not have been achieved had he not borne the reputation of being ready at any time to give or demand what was called satisfaction." *See:* Pilcher, J. E.: The Surgeon Generals of the United States Army. IX. Brevet Brigadier General Thomas Lawson, Surgeon General of the United [*sic*] Army, 1836-1861. Jour. Assoc. Mil. Surgeons, 14:405-410, 1904.

*Should be May, 1840.

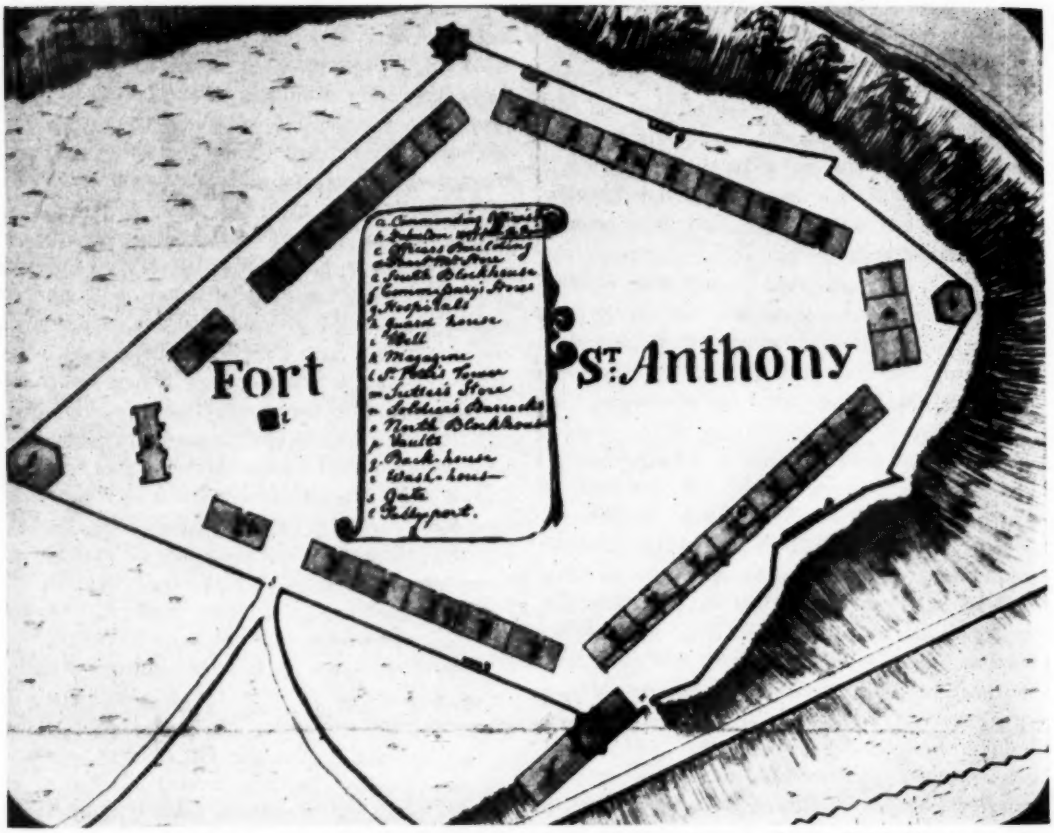

Fig. 2. Enlargement of a portion of a large folio plan of Fort Snelling drawn by Joseph E. Heckle at some time before 1825, in which year the name of the post was changed from Fort Saint Anthony to Fort Snelling, by recommendation of Major General Winfield Scott, approved by Secretary of War John C. Calhoun. A stone hospital was built at Fort Snelling in 1830, replacing the one designated in this map. Map reproduced by courtesy of the Army Branch, Quartermaster's Records, War Records Division, the National Archives, Washington, D. C.

the roof of the Portico, exposed to the external air—"[57] This, Surgeon General Lawson evidently concluded, was a flagitious wrong. The barometer, he pointed out to Dr. Turner, "should be kept 'in an *apartment* having an equable temperature.' The temperature of the open air at Fort Snelling is by no means *equable;* your Surgery therefore would probably be a more suitable, and certainly a more convenient location for it."[30]

The Laocoön's snare of governmental regulations enmeshed Dr. Turner again in 1844, when the enlistment of his hospital steward, Joseph Reed, of the First Infantry, expired. Turner valued this soldier's efforts highly, and Reed himself was willing to reënlist. A number of difficulties presented themselves. First, there was no recruiting officer at or near Fort Snelling. Second, there were already more soldiers at Fort Snelling than the prescribed standards allowed, which would appear to bar Private Reed's reënlistment. Third, if Private Reed were allowed to reënlist at Fort Snelling, would he be required to change his station when the station of his company was changed?[59]

". . . seeing that it is particularly important at a station so remote as this where the Medical Officer has little or no opportunity for selection, to secure the services of a well tried, habitually temperate, intelligent and faithful Steward," Turner wrote, "I would respectfully suggest if it meets your approbation that the necessary authority be obtained from Gen[l] H[d]. Quarters for re-enlisting the above named individual as Hospital Steward at Fort Snelling."[59]

It was, apparently, a dilemma too ponderous for Surgeon General Lawson to grapple with, for he replied to Dr. Turner:

"Your communication of the 4th Instant upon the subject of the re-enlistment of your Hospital Steward, has been received. The letter has been referred to the Adjutant General's Office, for the consideration and action of the General-in-Chief."[32*]

How the matter was concluded the correspondence does not show.

Yet, if Dr. Turner's efforts seemed faulty to Surgeon General Lawson, they certainly were esteemed by the few white settlers in the area. The Rev. Lucian Galtier (1811-1866), who came to the region in 1840, recalled that in August of that year he had been attacked by "bilious fever and ague."** He said he lay prostrate in the post hospital at Fort Snelling for two months, and that he "could not have recovered, were it not for the skill of Dr. TURNER, and the continued and kind attentions of his good lady."[18] Sibley recalled that when, in 1845, the senior Little Crow, chief of the Sioux at Kaposia village, accidentally shot himself, he (Sibley) asked Dr. Turner to go with him to attend the stricken warrior.[45] Dr. Turner did so, and when he announced that the wound would be fatal, the old chief smilingly agreed with the diagnosis. Both men were correct.

On another occasion, however, Dr. Turner's services seem not to have been valued by the Indians. A Dakota brave with the incredible name of Napehshneedoota *(Red man who flees not)* had fallen ill of a fever at Kaposia village; Dr. Turner's assistance was solicited. He sent some medicine to the Indian's tribesmen. The fever shortly thereafter subsided, but the unimpressed Indians maintained that their own medicine man could have cured the afflicted one in far less time.[67]

In 1845 a man wrote to Franklin Steele* (1813-1880), then sutler at Fort Snelling, from as great a distance as Leech Lake, saying that he had written to Dr. Turner for medicines, adding that the man who delivered his letter "will not be able to speak anything but Indian."[26]

Armstrong[1] wrote that Dr. Turner treated A. L. Larpenteur (1823-1919), then an interpreter for Indian traders, for typhoid fever in 1847 and 1848. When Dr. Turner left the fort on leave, Dr. Thomas S. Williamson (1800-1879), the Presbyterian missionary to the Sioux, apparently was paid to take his place. The Sibley Papers contain a document concluded on April 28, 1843, in which Dr. Turner agreed to pay Dr. Williamson "sixty dollars per month for your services as Physician for the command at Fort Snelling during my absence." On May 16, 1843, Dr. Turner advised Surgeon General Lawson that his medicines, instruments and hospital stores had been turned over to Dr. Williamson.[60] In the same year, Armstrong[1] noted, Dr. Williamson set down: "For attending on the Garrison and Indians for Dr. Turner, three months and eight days, $229.00. For private practice while living near Fort Snelling, $110.06."

Another curious aspect of Dr. Turner's interests while he was at Fort Snelling is mentioned in the diary of a settler who had a farm on the Mississippi across the river from the Kaposia village; in April, 1846, he recorded, Dr. Turner

*Major General Winfield Scott (1786-1866).

**An interesting note on malaria and his treatment of it was provided by Dr. Turner in his first year at Fort Snelling. In a communication to the surgeon general dated September 30, 1840, he wrote: "A large proportion of the cases of intermittent and remittent fever reported [from Fort Snelling] had their origin at Fort Crawford [Prairie du Chien, Wisconsin], and occurred here in men belonging to a company of infantry which returned to this post. There was nothing peculiar in the treatment of these cases, except, perhaps, the use of tartrate of antimony in combination with the sulphate of quinine, which was always prescribed when there was a reasonable doubt as to the condition of the system to bear the stimulating effect of quinine; and especially in the remittent cases, in which the diaphoretic effect of the tartrate of antimony was almost always produced when combined with the quinine, while the latter had its specific effect in preventing the recurrence of the periodic paroxysm. The following is the usual prescription: Sulph: quiniae, grs. x; tart: ant: et potass, gr. 1.; ft: pil: no. viij; one every hour during apyrexia. I consider the combination invaluable in the treatment of intermittents and remittents, but particularly the latter; and my conclusion is founded on my experience in the management of these diseases in Florida." *See:* Statistical Report of the Sickness and Mortality in the Army of the United States, Compiled from the Records of the Surgeon General's Office; embracing a Period of Sixteen Years, from January, 1839, to January, 1855. Washington, A. O. P. Nicholson, Printer [34th Congress, 1st Session, Senate Executive Document No. 96], pp. 77-78.

*Franklin Steele (1813-1880), who became sutler at Fort Snelling in 1838, bought the entire military reservation of that name, embracing more than 7,000 acres, from the Secretary of War for $90,000 on July 2, 1857, although he did not receive the deed until 1871. All the factors in this very curious transaction, which at the beginning was carried on with great secrecy, have never been satisfactorily explained. The deed conveyed only 6,394.80 acres to him because, in the interim, 1857 to 1871, the government had decided to retain 1,520 acres and the buildings of the fort. In the correspondence between Dr. George F. Turner and the surgeon general between 1840 and 1848 Dr. Turner several times mentioned the United States taxes he had collected from Franklin Steele, presumably levied on Steele's activities as sutler at the fort. *See:* Folwell, W. W.: A History of Minnesota. St. Paul, Minnesota Historical Society, 1921, vol. 1, pp. 432-434; 503-515.

had "Requested me to catch him 2 or 3 Gophers."[33]

Of Dr. Turner's relations with Dr. John Emerson (?-1844), owner of the celebrated Negro slave, Dred Scott,[21] nothing is known save a cryptic sentence in a letter written by Emerson's widow[12] on March 3, 1844, to H. H. Sibley: "As I supposed there was no such feeling [amity] for him [Emerson] by any one at St. Peters—for I know how bitter Dr. Turner & the Chaplain have been & how much they have done to injure him & any expression of friendship for him I have lost affords me some comfort." Nothing in Dr. Turner's letters amplifies Mrs. Emerson's complaints to General Sibley. A Bostonian, Turner may have been an abolitionist, but that is only conjecture.

Curiously enough, Dr. Turner's communications to the surgeon general disclose a short tour of duty that does not appear in the records of the Adjutant General's Office. On September 9, 1846, he sent to the surgeon general a return of instruments and stores which he had just placed in the hands of Assistant Surgeon Robert S. Holmes at Fort Snelling.[61] The next report is dated at Camargo, Mexico, December 5, 1846.[62] He had arrived there on November 24, 1846,[63] where, according to Duncan,[10] he succeeded Surgeon John B. Wells as purveyor of medical stores to General Zachary Taylor's army. Camargo, which Josiah Gregg[17] a month after Turner left it described as "the most important military depot on this line,"* in September, 1846, had become the purveying depot for Taylor's army. Prior to that, the purveying depot had been located at Point Isabel on the Rio Grande.[10]

At Camargo wretched staff work visited new miseries upon Dr. Turner: he received Army goods consigned to quartermasters at such preposterous destinations as Fulton, Arkansas, and Memphis, Tennessee;[63] he had to hire two civilian physicians to minister to Kentucky volunteers whose regimental surgeons were unaccountably absent, and was then queried by the acting surgeon general in Washington as to where the missing regimental surgeons were![25] "Considering the means at my control of performing the duties of this office," the harassed Turner wrote, "it is impossible for me to comply strictly & promptly with the regulations. I am constantly occupied in filling the requisitions of medical officers from every portion of the Army of occupation."[63]

Dr. Turner's tribulations in Mexico are not surprising, in view of Garrison's judgment:[19]

> The Mexican War (1846-48) is highly instructive to Americans as a campaign of small dimensions, entered upon caravan-wise with boyish, meridional enthusiasm, waged in an unknown country without forethought or adequate preparations, by an army equipped on a peace footing, and carried to a successful issue in spite of untold suffering from lack of clothing, supplies, rolling stock and adequate medical administration.*

Dr. Turner left Camargo on March 7[50] and arrived at Fort Snelling on May 18, 1847.[51] Nothing of moment appears in his letters to the surgeon general for the rest of that year; but on December 20, 1847, the first two instances of scarlet fever broke out, killing the two children concerned in a very short time.[47] The disease and its sequelae attacked thirty-seven children, four women and one man at the fort, taking the lives of six. It is interesting to observe that Turner believed the Sioux Indians, who he said had experienced epidemics of the disease twice previously, had developed a resistance to it.[47]

On July 31, 1848, Dr. Turner turned over his supplies and instruments to Assistant Surgeon Joel Martin (?-1854), his successor at Fort Snelling.[52] He had been ordered to San Francisco.[9]

After eight lonely years in the remote fastness of Fort Snelling and fifteen years' service in the Regular Army, his pay was still inconsiderable, even measured by the standards of the times. A surgeon of less than ten years' service received $60 a month; his subsistence amounted to four rations a day or the monetary value of $24 a month, one horse (value of $8 a month), two

*Because the town of Camargo, in northeastern Mexico, 4 miles up the San Juan River from where that river joins the Rio Grande, could be reached by steamboat if rains were heavy enough. In 1846 the San Juan River reached a height of more than 50 feet above low-water mark, and Army steamboats laden with men and stores easily reached that town from the town of Matamoros. *See:* Porter, J. B.: Medical and Surgical Notes of Campaigns in the War with Mexico, during the years 1845, 1846, 1847, and 1848. Am. J. M. Sc. 23: 13-37 (Jan.) 1852.

*Smith, the great authority on the War with Mexico, determined that of the volunteer soldiers, only 59,000 actually saw active service. Of each 100 volunteer soldiers, disease killed twelve; of each 100 regular soldiers, disease killed eight. Of each 100 volunteer soldiers, one was killed in battle or died of wounds; of each 100 regular soldiers, three were killed in battle or died of wounds. *See:* Smith, J. H.: The War with Mexico. New York, the Macmillan Co., 2:318-319; 511-512, 1919.

servants or the monetary value of $33 a month (they had the pay, subsistence and clothing of a private soldier), or a total of $125 a month.[41] Turner, with more than ten years' service, would have been paid somewhat more, but not much more.

During these tedious and no doubt frustrate years at Fort Snelling, Dr. Turner's chief delight may have been his daughter, born on December 22, 1841.[64] Possibly she was born at the fort, although there is nothing to indicate that such was the case. All that is known of her is that she perished tragically with her father, a dozen years later.

Dr. Turner was stationed at San Francisco until August, 1850.[9] He then served at various posts in Texas; in June, 1851, in his quarterly report to the surgeon general, he included a description of the town of Austin, and seemed to be well pleased with both the country and the climate.[46] In October, 1854, he was stationed at Corpus Christi, Texas.[9] A month later a newspaper in Boston published this melancholy note:[6]

> TEXAS. *Corpus Christi, Oct. 28.* The sickness of the South, which in former years has spared us, has this year not been so partial in our favor; a good many have been sick, and a few have died. Among the latter, Dr. Turner (surgeon of the U. S. Army), and his daughter, a beautiful young lady of twelve years, and a servant girl in the same family, have died. . . .

Dr. Turner died of dengue fever at Corpus Christi on October 17, 1854.[35,68] The young daughter of whom he had written[64] so proudly to General Sibley in 1842 apparently died with him.

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President's Letter

IS BUTTER BETTER?

Some time ago, the dairy industry was making use of a slogan that, in my opinion, was very appealing. The slogan was "It's Better with Butter." Immediately the imagination was stimulated to familiar pictures of crusty slices of bread, with rich, golden topping, or stacks of hot, tender wheatcakes, drenched with a flood of melted butter.

The country cooking that most of us have sampled at one time or another in our youth was based on recipes that called unabashedly, for a cup of butter, a half dozen eggs, generous amounts of heavy cream, and the like.

Hearty, nutritious foods are a tradition in this country; we have glorified in an abundance of good things for the table, and, not incidentally, have developed into the healthiest nation in the world—with notable progress in fields of both physical and mental achievement.

As a physician, I take deep pride in the health we enjoy and I look toward even greater strides in healthy living, advances which may not be completed in my lifetime, but which are being presaged with increased clarity each day.

For reasons which are, then, partly scientific—the contribution that natural dairy food products have made to health through good nutrition—and partly because of a personal regard for the esthetic appeal of these wholesome foods which have formed a background of culinary and even cultural traditions—for these reasons I am extremely doubtful of the ultimate success of the intermittent efforts of various individuals and groups to crowd butter off the American table with a substitute.

The decision of butter vs. any substitute is one that will be made by the American people, and it is my opinion, and the opinion of many of my friends and associates in the medical profession, that nothing can nor will take the place of butter.

J. F. Norman

President, Minnesota State Medical Association

Editorial

CARL B. DRAKE, M.D., *Editor;* GEORGE EARL, M.D., HENRY L. ULRICH, M.D., *Associate Editors*

DIABETES DETECTION WEEK —NOVEMBER 11-17

WHILE the discovery of insulin has been invaluable in the treatment of diabetes, the disease still deserves serious consideration, for it is on the increase and ranks eighth as a cause of death. Unknown cases of diabetes should be recognized so that advantage may be taken of modern treatment.

This is the purpose of Diabetes Detection Week —a nation-wide drive sponsored by the American Diabetes Association. The Detection Week spearheads the year-round effort to uncover cases of diabetes and to spread information about the disease. The Diabetes Detection Week Committee, to which Dr. Moses Barron of Minneapolis has just been appointed, has supervision of this particular activity of the association.

Some twenty-eight state medical associations and over 500 county medical societies have established diabetes committees. All state associations and every county society should have such committees.

What can physicians do to co-operate with the American Diabetes Association? In the first place, they can test each patient and members of their families for glycosuria. This should be done at least once a year. They can urge the appointment of a committee in their country medical society to carry on locally the detection and publicity work in their locality.

The principle of self-testing was approved by the American Diabetes Association as far back as June 5, 1949, and by the AMA House of Delegates on June 28, 1950. The Clinitest or Galatest is so simple that the convenience of home-testing seems to justify the approval of self-testing in this instance. Ninety minutes after a meal is the most likely time to detect a mild case of diabetes.

Minnesota is one of the twenty-eight states that have a state committee on diabetes. Dr. John R. Meade is chairman of the committee. Six county medical societies in the state will co-operate in putting on a detection drive during the designated week.

While each succeeding drive the past three years has been more successful than its predecessor, local success depends on local endeavor. Last year the city of Virginia, Minnesota, set the example for the United States in having the most successful drive of any city in the country.

With the co-operation of druggists, public health nurses and local press, a drive may be organized in any community. Literature is available at the headquarters of the American Diabetes Association, 11 West 42nd Street, New York 18, New York. The association will be glad to send pamphlets on organizing successful drives in a community.

If it is too late to organize a drive this year, start the ball rolling in your locality to put on an all-out drive next year.

SERUM HEPATITIS

A VERY illuminating presentation of the subject of hemologous serum jaundice or serum hepatitis was presented in the June issue of *What's New,* published by Abbott Laboratories.

A virus capable of producing a serum hepatitis can apparently remain viable in an immunized host for years. Injection of even a minute amount of such contaminated blood parenterally is capable of causing serum hepatitis in a matter of ten to twenty-one weeks following the injection.

The injection may be an intravenous transfusion of whole blood, plasma or serum. The greater the number of sources of pooled blood, the greater the chance of producing a serum hepatitis, for only infinitesimal amounts of infected blood are needed to produce the infection.

Intravenous injections of any sort for syphilis, diabetes, arthritis and the like, or parenteral injections of the antibiotics, vitamins or the like, for which a syringe or needle insufficiently sterilized after contaminated by blood from an infected individual can cause a serum hepatitis.

The experience of the United States army in

1942, in which some 28,585 cases of hepatitis and sixty-two deaths followed inoculations with yellow fever vaccine which was made with human serum, is recent enough to have left a vivid impression in the minds of many a physician who came in contact with the victims. The British army had a similar experience from the use of pooled mumps and measles convalescent plasma during World War II. As far back as 1920, Stokes and his co-workers at the Mayo Clinic reported seventy cases of "epidemic infectious jaundice" among 5,200 patients given antisyphiletic treatments during the preceding four years. In 70 per cent of the cases incubation was seven to twenty-three weeks.

Irradiation of blood is apparently not 100 per cent preventive, as demonstrated by four reports of cases of serum hepatitis following the use of irradiated plasma which appeared recently in the JAMA.

Certain conclusions from these observations may be drawn.

Since the virus is contained in the blood of the infected individual, thorough cleansing of syringe and needle is indicated. The mere cleaning by the use of alcohol of a lancet used to draw blood is definitely insufficient. Dipping in alcohol and flaming the lancet should assure safety. The boiling of needles and syringes for a few minutes is not 100 per cent preventive. Dry heat at 160° C. (320° F.) for an hour is the method recommended in the article mentioned. A prospective blood donor who has had jaundice of any type should not be used. This policy has been adopted by the Red Cross.

As serum hepatitis has been transmitted by accidental inoculation of infected blood through the broken skin, from a scratch or prick, care should be taken by laboratory technicians and physicians in the handling of blood or organs so as to avoid self-inoculation.

While the percentage of infected individuals is small, the danger of producing serum hepatitis by transfusion deserves due consideration. While the syringe is a less likely source of transmission of disease, yet it deserves consideration. In some hospitals a blood solvent, alcohol and ether are used successively before the syringe is autoclaved. Doubtless more careful cleansing of instruments contaminated by blood is advisable.

PHYSICIANS' INCOME IN 1949

THE survey of Physicians' Income undertaken by the Bureau of Medical Economics and the Office of Business Economics of the United States Department of Commerce was made public first in the July, 1951, issue of the *Survey of Current Business*, United States Department of Commerce. Dickinson and Bradley* presented an analysis of the survey which includes interesting data which merit mention at the risk of repetition.

Physicians may have been curious in the past as to the true status of the profession as a whole in the earning scale. This is the first survey to be made on a large enough scale to provide figures from which reliable conclusions may be drawn. The profession co-operated to the extent that over 55,000 of the 125,000 questionnaires sent out were filled in and returned. Some 30,000 were analyzed Figures for the interns and residents, military physicians and those who received most of their income from a medical school were excluded. The aim was to obtain figures for physicians in private practice dependent either on fees or salaries or both.

The average net income for all physicians in 1949 was $11,058. This figure is approximately 60 per cent of the gross income and represents the gross income minus the cost of practicing. In other words it costs $2.00 of every $5.00 collected to practice medicine. This income figure may seem low to specialists in their prime (forty to fifty years), and it may seem high to general practioners younger or older than this most productive decade in a physician's life. General practitioners, as is to be expected, average some $3,000 less and full specialists some $3,000 more than the general average. Salaried physicians average considerably less than independent ones, but they have certain compensations in the way of social security and retirement plans which partly at least make up for less income.

This average figure of $11,058 is some 150 to 170 per cent above the average income of $4,000 to $4,400 estimated for the years 1935-1939. This compared with the rise of 179 per cent per capita for the nation at large shows that the profession is just about keeping up with the inflationary trend since World War II—which is as it should

*Dickinson, Frank G., and Bradley, Charles G.: Survey of physicians' income. J.A.M.A., July 28, 1951.

be. It is interesting to note that this rise in income is not due to a 150 to 170 per cent increase in fees. According to the United States Bureau of Statistics, fees of physicians were only 38 per cent higher in 1949 than in the 1935-1939 period. The explanation according to the authors may be due to better collections and a greater output per physician because of more office and hospital work, increased technical assistance, better transportation and the like.

It is interesting to note further that the average income of physicians in 1949 was greater than that of the lawyers. The reverse was true in the twenties and thirties. The average for the dentists in 1948 was about $4,000 less than for the physicians in 1949 but showed an increase of 154 per cent, according to a survey published in January, 1950, by the United States Department of Commerce.

An interesting observation as to physician and income taxes is again brought out in this report. The accumulated investment of an intern in his seven or eight years of premedical and medical education is well over $30,000. A residency of three or four years adds thousands more. The years of returns from his investment are reduced because of his long period of education and training, and thus he will pay more income tax during his period of productivity than the individual who earns the same amount over a longer period of years.

Of further interest in the survey is the discovery that physicians in the far west—Arizona, Washington and California—do the best, while those in the New England states have the lowest incomes. In the group of salaried physicians Minnesota, North Dakota and New Hampshire are at the top of the list. We suspect the Mayo Clinic has some causative relation here.

As to the relation of the size of a municipality to income, those in towns of 1,000 or less in population have the lowest incomes and the incomes increase as the size of the cities increase up to the group of 250,000 to 500,000, above which they decrease. This suggests that cities of over 500,000 are probably over supplied with physicians, and this fact merits serious consideration in the selection of a location by young physicians. The well-known higher cost of practice the larger the city also undoubtedly has a bearing on this observation.

As to specialties, the neurological surgeons head the list, with the pathologists second. When it comes to salaried physicians, the roentgenologists head the list, with pathologists second.

According to the authors, the survey shows no general economic evidence of a national shortage of physicians such as is often voiced. If there were a shortage, the income of the profession would have outstripped the general percentage increase in all incomes incident to the inflationary trend since World War II.

MINNESOTA DEPARTMENT OF HEALTH

Additional Report of Death Associated With Pregnancy

During the past year the Maternal Mortality Committee of the Minnesota State Medical Association in co-operation with the Minnesota Department of Health has carried out an intensive and thorough study of each maternal death in Minnesota. The study included not only deaths due directly to pregnancy or the puerperium but also all female deaths where pregnancy was present but was not the cause of death and the postpartum period for three months following delivery. The study is now in its second year and it is planned to make this a continuing study, indefinitely, in an effort to still further reduce preventable maternal deaths.

Occasionally, pregnancy and the period of gestation or the postpartum period for three months is omitted from the death certificate when there is another specific cause of death. In such cases the Maternal Mortality Committee must use other means to identify such deaths. In order that the study may be as complete and as objective as possible it is important that reports of all deaths related to pregnancy or the puerperium should be made as promptly as possible to the Minnesota Department of Health. It is also important that the hospital, the physician, and the patient should be protected in the use of these confidential records.

In order that maternal deaths may be studied more promptly the State Board of Health has adopted the following regulation, effective July 20, 1951:

"Reg. 125. Any death associated with pregnancy (including abortion and extra-uterine pregnancy) or the puerperium (for a period of three months' postpartum), whether or not it is the actual cause of death, will be reported to the Minnesota Department of Health, Division of Maternal and Child Health by mail within three days after such date, by the attending physician and by the hospital where the death occurs.

"The following information will be included in the report:

Name and address of woman
Date of death Age at death
Cause of death
Name of attending physician and address
Name of hospital and address

"The above report is in addition to the usual report by death certificate to the Division of Vital Statistics as now required by law."

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

WELFARE STATE CALLED NON-REDUCIBLE

The dismal and inevitable course of socialism and the welfare state is still seen to vary little in countries where it has gained a foothold. Of the many nations of Europe where the scheme has been implanted, Norway is an example of the non-reducible quality of welfare state "service." According to Dr. William T. Bean, Principal, Butler (Pa.) High School, who is studying Europe's social services, "Norway's program . . . which is very extensive, has not been expanded; neither has it been reduced. . . . It would be almost impossible for any government to reduce the services at this point."

"Free Ride" Exposed

Dr. Bean warned about what Americans know is true of the socialist state—that "these non-reducible services, plus expenditures for defense, have produced a tax binder (shackle) and government control that are almost ruinous. . . ." Any socialistic service, like government medicine, becomes bound up so thoroughly in its own entanglements, that it is impossible to extricate it from disastrous government controls. Dr. Bean reports:

> "Government control is very extensive indeed. In addition to levying taxes, and thereby exercising a considerable measure of control, it has necessarily extended control over the lives of the people as it has extended services to them, and as it has taken over responsibility for their welfare. The people complain bitterly about the high taxes and government restrictions but they would never reduce the very thing that causes them. Obviously many people had expected these things to be done without any personal inconvenience to themselves. It is the old story of the 'free ride' exposed."

The proponents of the socialized state should learn of socialism's fallacies from the bitter experience of European nations. They have been repeatedly warned of the dangers involved in welfare state ideas, such as socialized medicine, but seem to continue to think that they themselves would be immune to the overpowering economic disadvantages that go with a socialized state.

Echoing this feeling, a recent issue of *Insurance Economic Surveys* has this to say about Dr. Bean's report:

> "If 20th Century 'Democratic' Socialism has proved one thing—it is that political ownership, management or control of the facilities of production, cannot succeed in a free society. It always reduces production of goods and services and thus lowers the living standard. Complete Socialism requires the enslavement of the people. It requires rigidly controlled and enforced labor. It destroys the last vestige of individual freedom. England and Norway and the other Socialist countries may someday get rid of their Socialist governments but their road ahead, under any circumstances, is not pretty. Socialism has infected their future. For the sake of our American employes and for the sake of all our children we must not let its disguised ideas take root here."

"CONTROL THROUGH PURSE" SCORED BY BRITISH M.D.

Although little has been heard lately from the British medical profession regarding their coping with the National Health Service, an occasional rumble of protest comes through, showing American doctors that the protests are very real in Britain, and that doctors throughout America should be constantly guarding their own cherished freedoms. Such a protest is reported in a current editorial in *Postgraduate Medicine*, wherein sound, logical reasons are presented for the doctors' objections to the scheme.

The editorial reports a lecture of Dr. T. T. Fox, who said that, in spite of some feeling that the health service had greatly enlarged the freedom of doctors by eliminating some of the commercialism found in the direct payment of bills by patients, he recognized that "payment of the profession from public funds could pave the way to

control of the physician by the administrators through the purse."

Control through the purse seems always to be the main foundation of socialistic schemes. Dr. Fox and other alert British doctors have found this to be tragically true and seem to have learned their lesson. But, they find themselves in a position where their hands are literally tied, for the entrenchment of socialism is deep and nearly immovable. British physicians should be commended for their attempt to cope with the situation bravely, but their courage and fortitude could well be directed to doing more about ridding themselves of socialist shackles, instead of merely putting up with them.

COST OF LIVING INCREASES BEYOND MEDICAL CARE COSTS

Complaints about high physician fees can be countered with the recently revealed 1950 Consumers' Price Index, which shows conclusively that the costs of medical care have not risen as rapidly as, nor in proportion to, the costs of general goods and services. Dr. Frank G. Dickinson, Director of the Bureau of Medical Economic Research of the American Medical Association, has analyzed this recent report, stating that it is considered "our best general measure of the purchasing power of the consumer's dollar."

Advances in medical care costs have not kept pace with those of the general cost of living—and that advantage has been passed on to the user of that service. The price of medical care has risen 48 per cent since the base period (1935-1939), while the general cost of living has risen 72 per cent. According to Dr. Dickinson's report, this means that a dollar in the base period could buy a dollar's worth of medical care or other goods and services, but a dollar in 1950 could buy only 58 cents' worth of all goods and services. However, this 1950 dollar could buy about 68 cents' worth of medical care.

Dr. Dickinson analyzes this small increase in the price of doctors' services:

"The relatively small increase in the price of physicians' services in an inflationary era is to be expected. These prices are influenced by custom and are not as sensitive to changes in the economy as are the prices of most goods and services—particularly those whose prices must cover the costs of many other items used in their production. Physicians' fees belong to a class known as 'sticky' prices; they move neither up nor down as rapidly or as far as other prices. The economic position of the physician, however, becomes adjusted to changes in the rest of the economy in other ways. Thus, while physicians' fees had not increased as much as other prices, by 1949 the physician's collections had become so much better and his 'output' so much greater that his over-all income had increased since 1935-1939 just about as much as the national income per capita. In order for the physician to retain the same economic position in a time of rapidly changing price levels, it is not necessary for him to change his price to the extent that other prices have changed."

SHEARON SCORES "PIECEMEAL SOCIALIZATION"

Marjorie Shearon has again lashed out against what she calls "piecemeal socialization" in the Federal government. Feeling that since the issue of outright socialization of medicine has been largely met and defeated, she warns that there are many forces still trying to socialize in small ways and gradually develop a completely socialistic form of government. One of these schemes is the bill for "free hospitalization insurance" for the aged, which would be brought within the scope of the Social Security bill.

Ewing objects to the term "free hospital insurance" because he says that workers would pay for it with their Social Security taxes, just as they "pay" for their old-age insurance. But, says Mrs. Shearon, "since the OASI system is not financially sound and since many workers pay only ten cents on the dollar for the benefits they receive, it is ridiculous for Ewing to talk about the OASI system 'paying' for the 'hospital insurance for the aged.' The Federal Security Agency invariably sells, or tries to sell, its programs and its bills by misrepresenting costs and the long-range, overall plan for setting up a completely Socialistic State."

Government Insurance Subsidized

Ewing has made his usual criticism of private insurance by citing "the failure of private hospitalization to meet adequately the needs of the persons who would be protected under the proposed program," and claiming that this failure "caused some persons to go without needed hospitalization." Mrs. Shearon's comment is: "Of course, if private insurance could be as heavily subsidized for adverse risk as is Government 'insurance' under the Social Security System, private

insurance could cover as many people as Ewing proposed to cover."

Another weak argument Ewing uses for his proposed plan is this statement: "Some get hospital services for which they or their relatives pay by sacrificing other essentials, depleting savings or going into debt."

And the statement is answered by Mrs. Shearon:

"From this we judge that Ewing disapproves of people paying their own way in the world. And what are savings for if not to be used for the proverbial rainy day? As for going into debt, there is more validity in assuming debt for a hospital bill than for a television set or other luxury. We mention these differences in viewpoint because we think Ewing's philosophy is once again demonstrated to be completely Socialistic. According to his way of thinking, no one should save, no one should pay his own bills, no one should assume personal responsibility for the things he needs in life. Instead, everyone should depend on the Government."

VOLUNTARY INSURANCE COVERS OVER HALF OF AMERICANS

The latest report of the Health Insurance Council shows that at least half of the nation's population at the end of last year was covered by one type or other of voluntary protection against the economic hazards of sickness and accident.

The report also revealed that all forms of voluntary health protection scored tremendous gains in 1950, to set new records. Hospital expense protection covers the largest number of people and was extended to nearly 80 million persons—17 per cent greater than the figure of the preceding year. The number of people protected against hospital costs has more than doubled since the end of World War II—attesting to the growing appreciation of voluntary insurance advantages.

Surgical and medical expense coverage also increased greatly. About 54 million persons had protection against surgical expense, which amounted to an increase of 32 per cent over the preceding year. A year-to-year gain of 28 per cent was recorded by medical expense protection which covered nearly 22 million persons in 1950.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Julian F. DuBois, M.D., Secretary
230 Lowry Medical Arts Building
Saint Paul, Minnesota

SAINT PAUL DENTIST CONVICTED OF ILLEGAL PRACTICE OF HEALING

Re. State of Minnesota vs. Earl S. Weber

On August 10, 1951, Earl S. Weber, fifty-five years of age, was sentenced by the Hon. Arthur A. Stewart, Judge of the District Court of Ramsey County, to pay a fine of $500 or to serve ninety days in the Ramsey County Jail, following a plea of guilty by Weber on August 7, 1951, to an information charging Weber with the crime of practicing healing without a basic science certificate. Weber paid the fine.

Weber, a licensed dentist, who maintained an office at 311 Newton Building, Saint Paul, was arrested on July 31, 1951, following a joint investigation by Saint Paul police officers and representatives of the Minnesota State Board of Medical Examiners. The investigation disclosed that a twenty-five-year-old married woman had gone to Weber for the purpose of having an abortion performed. Weber denied that he intended to perform an abortion but admitted that he made a vaginal examination of the patient, advised the patient she was pregnant and inserted "about 12 inches of gauze in her vagina." Weber was paid $50.00 for his services. The patient became ill and was hospitalized in a Saint Paul hospital.

Weber has a previous conviction of a felony, having entered a plea of guilty on January 27, 1949, in the District Court of Ramsey County to an information charging him with the crime of abortion. For that offense Weber was sentenced to a term of not to exceed four years in the State Prison, but the sentence was stayed and Weber placed on probation for a period of four years. His probationary period, however, was terminated on May 10, 1951. When legal counsel for Weber made a plea to the Court for leniency on the grounds that Weber is one of four Negro dentists in Saint Paul and that his dental services are in demand by the Negro population of this City, Judge Stewart pointed out that Weber had a prior conviction for abortion and became involved in another difficulty within three months from the time he was released from probation. Weber is a graduate of the Dental School of the University of Minnesota in 1921. He has surrendered his dental license and his annual registration card for cancellation.

MINNEAPOLIS MAN SENTENCED FOR SALE OF POLIZER DEVICE

Re. State of Minnesota vs. Otto W. Dressler

On September 17, 1951, Otto W. Dressler, seventy-four years of age, formerly of 204½ Tenth Street South, Minneapolis, was sentenced by the Hon. Harold N. Rogers to a term of six months in the Minneapolis Workhouse following a plea of guilty entered by Dressler on July 27, 1951, to an information charging him with the crime of practicing healing without a Basic Science certificate. Dressler was arrested on June 13, 1951, following a complaint being made by a Minneapolis man that Dressler had sold him a Polizer device as a cure for asthma. Dressler charged $41.50 for the device. The purchaser claimed that he received no benefit and made a complaint to the Minneapolis Police Department. The matter was referred to the Minnesota State Board of Medical Examiners following which Dressler was arrested. Dressler stated to the Court that while he still believed in his

device, he was no longer engaged in such work and had moved to Eau Claire, Wisconsin, where he is living with a son. The Court then stayed the execution of the sentence for two years on condition that Dressler does not, in any manner, violate the Minnesota Basic Science law or Medical Practice act. Dressler made restitution of the $41.50.

Dressler has a previous conviction in the United States District Court in Minneapolis, where he was found guilty on December 31, 1949, by Judge M. M. Joyce of "having unlawfully shipped in interstate commerce a certain device, to-wit: Polizer, in violation of the Federal Food, Drug and Cosmetic Act, in that said device was misbranded." For that offense Dressler, on June 12, 1950, paid a fine of $100. In that case the Government charged that the Polizer device was represented as being efficacious in the cure and treatment of over 60 ailments ranging from arthritis to heart trouble. The Government offered evidence that the device was misbranded because it had no therapeutic value whatsoever. Judge Joyce pointed out that expert witnesses of the highest rank in the State of Minnesota had examined and tested the Pol-izer device and found that it was without any therapeutic value and, therefore, useless in the treatment of any such ailments.

MINNEAPOLIS MAN SENTENCED FOR AIDING IN ABORTION CASES

Re State of Minnesota vs. John E. Capra

On September 13, 1951, John E. Capra, thirty-three years of age, 1500 Third Avenue South, Minneapolis, was sentenced by the Hon. Theodore B. Knudson, Judge of the District Court of Hennepin County, to a term of not to exceed four years in the State Prison at Stillwater, following a plea of guilty entered by Capra on August 15, 1951, to an information charging him with the crime of abortion. Capra, who holds no license to practice any form of healing in the State of Minnesota, was placed on probation by Judge Knudson for a period of three years. The Court warned Capra that any violation of his probation would result in his being committed to the State Prison.

Capra was arrested on May 8, 1951, by Minneapolis police officers following an investigation by representatives of the Minnesota State Board of Medical Examiners and police officers from Minneapolis and Saint Paul. That investigation disclosed that a twenty-two-year-old Benton County girl was seriously ill at a Saint Cloud hospital following a criminal abortion performed by one Frank H. Masterjohn in the room of the defendant Capra at 1500 Third Avenue South, Minneapolis. Capra, a bartender, admitted to the Court that he had sent seven to nine cases to Masterjohn for a similar purpose. Masterjohn was arrested at the same time and entered a plea of guilty on May 24, 1951, but was awaiting sentence at the time the case against the defendant Capra was completed in Court. Capra also stated that he received $25.00 from each of the first several abortion cases and $100, or more, from each of the last four or five abortion cases. According to Capra, the abortions were all performed by Masterjohn in the living quarters of the defendant Capra.

Despite the encouraging decline of the death rate, and new techniques which should be helpful in the future, tuberculosis is still one of the most important public health problems. It will take many many years of undiminished energy to reduce it to an unimportant level.—JAMES E. PERKINS, M.D., Transaction, 1950, Conference of Public Health Association, New York City.

PRIVATE HEALTH INSURANCE GAINS

The Health Insurance Council is made up of nine trade associations in the life and casualty insurance fields. The reports of the Council, therefore, should be reliable. Its fourth annual Survey of Accident and Health Coverage in the United States indicates that at least half of the nation's population is covered by some type of voluntary insurance against the hazard of sickness and accident.

Those protected against hospital expense had been increased to 76,961,000 at the close of 1950, an increase of 17 per cent over the figure of 66,044,000 the year before. The present number of insured is double the number insured at the end of World War II.

Protection against medical expense also increased by leaps and bounds in 1950. Insurance against surgical expense was provided to 54,477,000 persons by the end of 1950 which is an increase of 32 per cent over the figure of 41,143,000 covered at the beginning of the year. Insurance against medical expense, as distinguished from surgical expense, covered 21,589,000 persons, an increase of 28 per cent over the figure of 16,862,000 in 1949.

Disability or weekly indemnity which provides protection against loss of income due to disability also showed an increase of 9 per cent to include 37,293,000 persons which is 60 per cent of the employed civilian population entitled to such benefits.

These figures do not include individuals covered solely by compulsory government insurance but include the types of insurance provided by insurance companies, Blue Cross, Blue Shield, fraternal bodies, industries, medical societies, universities and other private organizations.

There is every reason to expect a still further increase in 1951. Just what percentage of the population must be covered before the proponents of compulsory health insurance, who have claimed that health insurance under private auspicies can never supply the need, will be convinced of their error?

Minnesota Academy of Medicine

Meeting of March 14, 1951

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, March 14, 1951. Dinner was served at 7 o'clock and the meeting was called to order by the Vice President, Dr. O. H. Wangensteen, in the absence of the President.

There were forty members and two guests present.

The scientific program followed.

HYPERTROPHIC PYLORIC STENOSIS

HAROLD F. FLANAGAN, M.D.

Dr. Harold F. Flanagan, of Saint Paul, read his Inagural Thesis on the above subject. Lantern slides were shown. (See page 957.)

AVASCULAR NECROSIS

With a Report of Cases Involving the Distal End of the Clavicle and Odontoid Process

EDWARD T. EVANS, M.D.

Dr. Edward T. Evans, of Minneapolis, read his Inaugural Thesis on the above subject. (See page 970).

The meeting was adjourned.

WALLACE P. RITCHIE, M.D., *Secretary*

Meeting of April 11, 1951

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, April 11, 1951. Dinner was served at 7 o'clock and the meeting was called to order at 8 p.m. by the President, Dr. W. H. Hengstler.

There were fifty-six members and two guests present.

Minutes of the March meeting were read and approved.

Dr. Martin Nordland read the following memorial to Dr. Gustav Schwyzer, and a motion was carried that this be spread on the Proceedings of the Academy and a copy sent to the widow.

GUSTAV SCHWYZER

Dr. Gustav Schwyzer, a member of this Academy, died suddenly at his winter home in Biloxi, Mississippi, February 2, at the age of eighty-three. He was born in Switzerland and educated to the profession of medicine in Europe. As a relatively young man, in the latter part of the last century, he came to America, bringing with him a knowledge of surgery which was far in advance of that practiced in this region at that time. His training in goiter surgery under Theodore Kocher made him a pioneer here in this field. He gave of his knowledge freely to his colleagues and earned their respect for his skill.

Dr. Schwyzer was a man of dignity, respected by all. He never sought the popularity of the crowds, but by those who knew him well was counted a beloved man in whom they could place confidence and trust. He was always loyal to those who became his friends. He displayed a full measure of sympathy toward his fellowmen. The wide extent of his charities is not known to the casual observer, but his generosity was deeply appreciated by scores of persons whom he befriended.

Dr. Schwyzer loved life and lived intensely. He was a man of great courage and energy. He channeled this knowledge, courage and energy to the welfare of his fellow man through his surgical work. His interest in his profession continued to the end. He loved the Academy and attended the last time in November, 1950.

His indomitable spirit was the admiration of all who knew him. Not only was this true for the friends in his adopted land, but the memory of his spirit has lingered in his native Switzerland where in February services were held in his honor. Never did this spirit bow to illness or adversity of any kind. It carried him courageously forward, and he lived life to its fullest until his sudden death on February 2, 1951.

He contributed many articles to the literature of goiter on which he was considered an authority. In Halstad's "Operative Story of Goiter," his name is mentioned in the first successful operative cases reported by Kocher in 1898.

He was a charter member of the Minneapolis Club, a member of the Founders Group of the American College of Surgeons, Western Surgical Association, the Minnesota Academy of Medicine, the American Medical Association, Minnesota State Medical Association and Hennepin County Medical Society. Besides his association with the Northwestern Hospital, he was also chief surgeon for many years at the Minneapolis General Hospital, and surgeon for the Minneapolis Street Railway and the Great Northern Railroad.

Dr. Schwyzer was characterized by his attention to discipline and his enthusiasm for his professional work.

He is survived by his wife, the former Agnes Jorgens, of Minneapolis, and one son, Werner, of Houston, Texas.

MARTIN NORDLAND, M.D.

The President appointed Dr. A. E. Benjamin to prepare a memorial to Dr. H. B. Sweetser, Sr., and Dr. Edward T. Evans to prepare a memorial to Dr. Vernon L. Hart, to be read at the next meeting.

The president reported that the Executive Committee had proposed the elevation of Dr. Wilder's name to the Honorary Membership list; Dr. Wilder is now in Washington, D. C. and has asked that he be placed on such membership list as is constitutionally possible. This proposal was carried.

The election of new members resulted in the naming of Dr. H. B. Sweetser, Jr., and Dr. Asher White as candidates for membership in the Academy. The Secretary is to notify them of their election.

The scientific program followed.

Dr. Thomas Lowry, of Minneapolis, read his Inaugural Thesis. Lantern slides and x-rays were shown.

THE QUESTION OF CARDIAC TRAUMA

THOMAS LOWRY, M.D.
Minneapolis, Minnesota

The subject of injury to the heart muscle is a large one. Clearly, every variety of cardiac disease "injures" the heart in one way or another. I am sure that most of us who deal with heart disease are accustomed to employ loosely the phrases "myocardial injury" and "myocardial damage" when referring to the effects of such widely different pathological processes and agents as the anoxia of coronary insufficiency, the effects of rheumatic infection, and the myocarditis produced by diphtheria toxin.

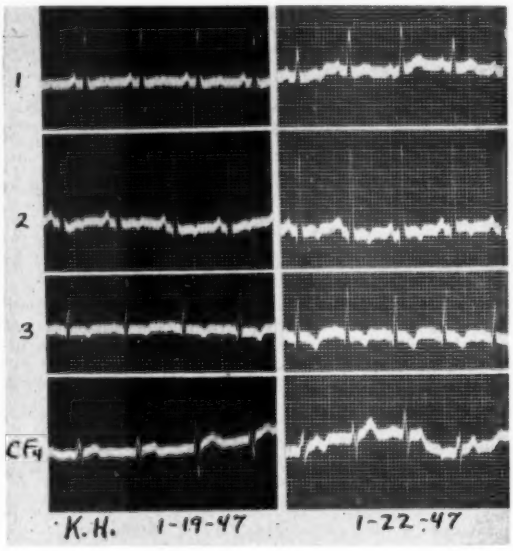

Fig. 1. K.H. 1-19-47 (four days after injury): T1 low and diphasic, T2 diphasic to negative, T3 negative, T4 low and tending to be diphasic. 1-22-47: T1 now more positive but T2 and T3 distinctly more negative.

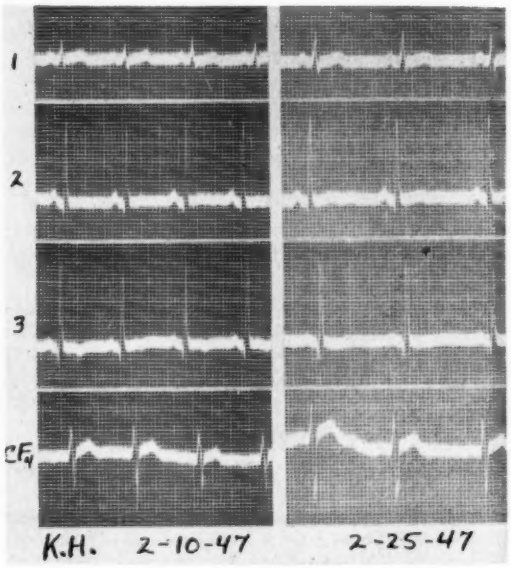

Fig. 2. K.H. 2-10-47 (about four weeks after injury and clinically much improved): T1 positive, T2 isoelectric, T3 negative but less so than in previous tracing. 2-25-47: T1 and T2 now positive and T3 isoelectric.

The present discussion, however, will be limited to consideration of non-penetrating cardiac trauma resulting from physical violence. It is proposed, for the purpose of this paper, to omit the condition of so-called cardiac "over strain," which some authors have included as a type of trauma. It is manifestly impossible, under a given set of circumstances, to say when the strain on a heart becomes excessive, and in any case, damage which might be caused by such means would be in no way comparable to injury by physical violence.

My interest in the question of cardiac trauma was aroused by problems presented in the care of two patients whom I had the opportunity to study about four years ago, and whose case histories I should like to outline.

K. H., a twenty-five-year-old salesman, sustained a severe blow to his left posterior chest in an automobile accident January 15, 1947 and was first treated at the Minneapolis General Hospital. On January 18, 1947, he was transferred to Northwestern Hospital where he arrived in rather poor condition. The more important injuries were: fractures of the left scapula and of ribs three through ten on the left side. Hemopneumothorax was present on the left, and there was marked subcutaneous emphysema. Other findings on examination were: temperature 99, pulse 96, respirations 26, blood pressure 210/150. The patient had a pale gray cyanosis, was cold, sweating, and dyspneic, with shallow painful breathing. The heart sounds were somewhat distant and blurred, with a "tic-tac" quality. There was evidence of pneumothorax on the left; the right lung was clear except for a few scattered moist râles.

A few hours after admission, the patient's condition became alarming. He developed a steady boring precordial pain, radiating through to the back and associated with severe aching in the left shoulder and upper arm. The pain increased progressively in severity and did not respond to codeine, demerol or morphine. At the same time, dyspnea increased and the heart rate and respiratory rate rose; the blood pressure remained high. When this paroxysm of pain, which lasted 5 or 6 hours, was at its height, the temperature was 102.4°, the heart rate 136 with regular rhythm, the respiratory rate 60, and the blood pressure 200/120.

Combined use of morphine and oxygen finally controlled the pain and it was felt that the oxygen had more effect than the morphine. The following day a pericardial friction rub was heard.

The man had a stormy course with high fever, rapid heart rate, and fluctuating blood pressure, which eventually stabilized in the normal range. About ten days after

admission, marked improvement took place and from that time on, his recovery was uneventful. He was considered to have a rather severe myocardial contusion and was kept in bed six weeks. His electrocardiograms appear in Figures 1, 2 and 3.

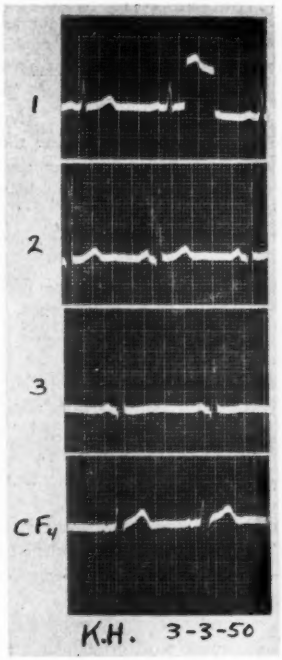

Fig. 3. K.H. 3-3-50 (three years after the injury): The electrocardiogram which became normal three months after the injury has shown essentially no changes since that time and still remains a normal tracing.

In this case, the chief abnormalities are in the T waves. The successive changes of pattern indicate the acute nature of this process. It is noteworthy that this electrocardiogram remained abnormal up to the time of the patient's discharge from the hospital. Later follow-up (Fig. 3) showed the return of a normal pattern.

G. R., a forty-nine-year-old steam fitter was admitted to Northwestern Hospital by transfer from another hospital two days after an automobile accident. In addition to a head injury and a compound fracture of the right leg, there were fractures of ribs 2 through 5 on the right and severe contusions of the thorax. The patient was in poor condition, with temperature 101.2°, pulse 90, blood pressure 170/120, respirations 24. A right hemopneumothorax was found, and subcutaneous emphysema was present. The heart size was normal, but the sounds were distant, with many extrasystoles. There was no friction rub and no murmur.

The electrocardiograms are of interest (Fig. 4). The first tracing, made February 1, 1947, soon after admission and 4 days after the injury, shows abnormal P waves especially in leads 2 and 3, suggesting auricular damage. There is also slurring of the QRS complexes and the T waves in leads 1, 2, and 3 are abnormal. The second tracing was made February 11, ten days after the first and indicates a rapid change in the direction of normal. Unfortunately, the patient died suddenly on February 17 of a large pulmonary embolus.

At autopsy the pericardial sac was obliterated by "moderately firm fibrinous and fibrous adhesions." The heart weighed 425 grams, and moderate dilatation of the

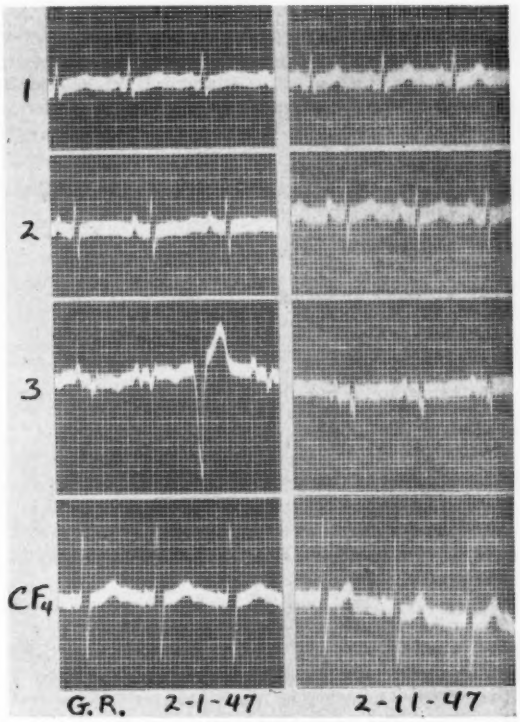

Fig. 4. G.R. 2-1-47 (four days after injury): P2 high and peaked. P3 diphasic. T1 low, T2 low and diphasic, T3 negative. A ventricular extrasystole is seen in lead three. 2-11-47 (Two weeks after injury): The pattern is returning toward normal with T1 positive, T2 positive, T3 much less negative; the abnormalities of the P waves are less striking.

right atrium and right ventricle were considered secondary to extensive thrombosis in the pulmonary arterial tree.

Microscopically the heart showed "inflammatory reaction in the epicardium; some increased vascularity and edema in the heart muscle."

This patient had survived his cardiac injury and was apparently making a good recovery when his pulmonary embolism occurred. The autopsy indicated that the heart had been damaged, superficially at least, but there was no evidence to suggest that this man's death was related to his cardiac injury or that *he would necessarily have had any cardiac disability, had he survived.*

As we consider this subject of cardiac trauma, a number of questions arise concerning its various phases. The remainder of this discussion is an effort to find answers to the more important of those questions. In this quest I have reviewed the more recent pertinent literature and have also studied the case records of patients admitted to the Minneapolis General Hospital because of thoracic injuries.

In the first place then, what are the effects of injury upon cardiac structure and function? The general types of injury to the heart can be put in three categories:

1. Direct or contre-coup blow, resulting in surface contusion.
2. Compression between sternum and vertebral bodies in a crushing injury, often resulting in endocardial tears.
3. Violent strain upon the attachments of the heart as a result of rapid deceleration in "high speed accidents," resulting in ecchymosis of the auricles near the orifices of the great veins.

The known effects of trauma upon the heart can also be listed rather briefly. They are:

1. Pericardial laceration or contusion
2. Extrasystoles
3. Auricular fibrillation
4. Partial or complete heart block
5. Damage to the myocardium shown by electrocardiographic changes only.
6. Injury to the mitral or aortic valve, leading abruptly to cardiac failure.
7. Immediate cardiac rupture, with sudden death.

These, then, are the types of injury encountered, and their usual cardiac effects. Next we may well ask how often the heart sustains an injury which is clinically detectable. On this point, evidence from the literature becomes rather confusing.

In the first place, most of the papers reporting series of cardiac injuries are clearly attempting to report a *large* number of cardiac injuries, and the minute electrocardiographic changes which are shown as evidence to prove such lesions are in many instances entirely unacceptable. Other reports are based upon findings in patients first examined many weeks or months after their alleged injuries, and in these, conclusions as to causation of symptoms and signs by remote trauma seem quite unwarranted.

However, Barber in 1944 reported on seventy-five patients who, after thoracic injuries, had routine electrocardiograms. Of these, twenty (26 per cent) showed some slight but definite abnormalities within forty-eight hours of the accident. Two of the patients died—of their other injuries. One of these was found to have bruising of the right auricle. The other, who died of pneumonia, showed no evidence of a cardiac lesion at autopsy; this death occurred 12 days after an electrocardiogram had shown distinct abnormality, presumably allowing time for healing of a bruise or other minor lesion of the heart muscle. From the small number of careful and unbiased reports, one gains the impression that around 25 to 40 per cent of patients suffering chest injuries can be shown to have electrocardiographic abnormalities which undergo rapid changes and soon revert to normal. Such evidence strongly suggests that myocardial trauma occurs rather frequently in cases of chest injury but is almost invariably slight and quick to heal—except in those individuals who succumb within a few hours after an injury.

Corroborative information is gained from the files of the Minneapolis General Hospital, where, since the first part of 1950, an attempt has been made to secure electrocardiograms routinely in all cases of injury to the thorax.

About fifty recent case records were examined, of which only fifteen were found to contain sufficient electrocardiographic data to be suitable for analysis. For a diagnosis of cardiac injury, it was required that there should be definite evidence of myocardial damage found by electrocardiogram within forty-eight hours after the injury, and that the findings should change distinctly within the subsequent week, confirming the acute nature of the process. Three patients between the ages of fifty-five and seventy were considered to have minor electrocardiographic abnormalities due to hypertensive or arteriosclerotic heart disease, rather than to their injuries.

Five of the fifteen cases (33 per cent) had acceptable evidence of myocardial injury. Of these, three showed changes limited to the ST segments and T waves. One developed, in addition, widening of the QRS complex. The fifth had first degree heart block and transient bigeminal rhythm, with many ventricular extrasystoles.

Of these five patients, three died, and autopsies showed the causes of death to be unrelated to the cardiac injury (one head injury, one gastric hemorrhage, one retroperitoneal hemorrhage). In one of the fatal cases, myocardial bruising could be demonstrated at necropsy. The other two had no pathologic evidence of heart damage, and in both these patients the electrocardiogram had returned to normal before death.

These results, drawn from a very small number of cases, are in agreement with previously reported figures. They confirm the impression that demonstrable cardiac damage occurs in about one-third of patients sustaining injuries to the thorax which require hospitalization.

What then are the symptoms, physical signs, and electrocardiographic changes which point to a diagnosis of cardiac contusion? The most important ones are as follows:

SYMPTOMS

1. Precordial pain, often with characteristic cardiac radiation to the jaw, left arm, back, etc. In some cases there is also pain on swallowing.
2. Dyspnea and orthopnea.
3. Cyanosis.
4. Evidences of shock: gray pallor, sweating, weakness, restlessness, etc.

PHYSICAL SIGNS

1. Cardiac arrhythmias of all sorts.
2. Fluctuations of blood pressure. These may be either upward or downward.
3. Increase in the area of cardiac dullness, (suggesting pericardial effusion or myocardial dilatation).
4. Paradoxical pulse.
5. Abnormal heart sounds.
6. Pericardial friction rub.
7. Cardiac murmurs, if they appear suddenly or change abruptly.
8. Signs of cardiac failure, such as pulmonary edema or hepatic passive congestion.

The usual electrocardiographic abnormalities encountered are these:

1. Extrasystoles (common).
2. Auricular fibrillation.
3. Auricular flutter and other disorders of rhythm (rare).
4. Abnormal P waves.
5. Changes in the QRS complex.
6. Abnormalities of the ST segment and T wave.

In order to ascribe any of these alterations in the electrocardiogram to recent cardiac injury, one must be able to demonstrate definite, and fairly rapid *changes* of the pattern occurring under observation.

Once detected, what is the proper course of treatment to adopt for myocardial injury? On this point, there can be little argument. It seems entirely logical to treat myocardial trauma in the same general manner as myocardial infarction. All who express any opinion on the subject agree with this point of view. It is probably true that such a policy will in many cases be unnecessarily conservative, but where we are dealing with a definite cardiac lesion, we can afford to be conservative.

Thus, the mild injury, which shows itself only through transient abnormality in the electrocardiogram, should be cause for keeping a patient in bed three or four weeks. More severe lesions indicate more prolonged treatment. Clearly, if complications such as arrhythmia or congestive heart failure supervene, appropriate therapy, with digitalis, quinidine, oxygen, diuretics, etc., should be employed just as if the same events had occurred in the course of a myocardial infarction.

The prognosis of cardiac injuries, as has already been suggested, is generally good. If the victim survives the immediate effects of the injury, he is not apt to die because of his cardiac damage. There are, of course, exceptions to this statement. In a small number of cases, cardiac failure develops after an interval of two days or more following the injury. This may prove intractable and lead to a fatal outcome. Then there is the rather rare accident of delayed cardiac rupture. This event, usually occurring during the second week after injury, has been found to follow an endocardial tear which extends outward into the myocardium, and therefore fails to heal like an ordinary surface contusion.

Granted that those who survive the immediate effects of an injury to the heart are unlikely to *succumb* after an interval, what of disability and symptoms related to the heart in these individuals? Having gone through much of the available published opinion, and tried to weigh the evidence, I have reached the conclusion that disability resulting from cardiac trauma, though it does occur, is a rarity. The literature contains almost no examples in which the evidence is acceptable. Some patients may have twinges of precordial pain from time to time, which can be presumed to arise from pericardial or pleuropericardial adhesions. This symptom is annoying but not disabling, and it is not of serious prognostic import.

It is interesting to note that several authors have published papers which purport to prove the occurrence of cardiac symptoms resulting from trauma. In at least one such communication, the outcome of the litigation (generally an award of compensation) is appended to each case report. Careful analysis of such records reveals, in most of them, a surprising lack of adequate evidence in support of the conclusions which are drawn. For the most part, these case reports describe symptoms such as angina pectoris or mild cardiac failure, which the patient is able to date from a blow on the chest. In many published instances, cardiac examination has been first done two months or more after the alleged injury. The fallacy of such reasoning is too obvious to require further comment.

It goes without saying that the prognosis and management of any given case must be decided on its own merits and not according to any rule, but it is cheering to know that virtually none of these patients develop any serious symptoms or disability, once the immediate post-traumatic period is safely past.

This knowledge leads to one other conclusion which, in closing, I should like to emphasize. Though remaining on the alert for cardiac contusions in individuals who have sustained chest injuries, one must scrupulously avoid giving these patients cause for unnecessary worry. In a severe blow on the chest there is excellent building material for a disabling cardiac neurosis. The physician must be, if anything, more careful to avoid scaring his patient than he is to avoid overlooking a minor myocardial injury.

Summary

1. The subject of non-penetrating injuries to the heart has been reviewed, and illustrative case material has been presented.

2. Mild cardiac injuries are probably relatively frequent.

3. Efforts should be made to detect myocardial damage in individuals suffering thoracic trauma; and patients sustaining such damage should be conservatively treated.

4. The prognosis of mild myocardial injury is good; overemphasis on the seriousness of the condition may cause patients unnecessary alarm and lead not only to neuroses, but to unwarranted compensation claims.

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DISCUSSION

DR. CARL B. DRAKE, Saint Paul: I am reminded of a patient who was admitted to the Ancker Hospital in the 1920's. This man in his thirties had been leading a heifer by means of a rope in the stock yards in South Saint Paul. The heifer became obstreperous and a twist was taken on a post of a gate. The heifer reared and the gate flew open and struck the man a severe blow on the chest. He was knocked down and pretty well out but had enough strength to drag himself through the gate and out of harm's way. After a brief period of time he was able to walk to the office of the company doctor but instead of waiting he went home. The following day he was admitted to the Ancker Hospital. His heart was not enlarged but it was fibrillating. The one blood pressure reading recorded was something like 140 systolic. There was no decompensation. The fibrillation resisted quinidine administration and he left the hospital after a few months. An electrocardiogram taken at the Miller Hospital confirmed the presence of a fibrillation. A few months later this man returned to the Ancker Hospital with marked enlargement of the heart and severe decompensation. The heart was still fibrillating. He died a short time later.

The question arose as to whether the heart had sustained an injury from the severe blow on the chest and whether the fibrillation was due to the blow. Workman's compensation was involved.

At autopsy, no evidence of heart injury was found but the heart was markedly enlarged. Hypertension was reported as the cause of death although there was no history of hypertension. The microscopic kidney findings I do not recollect, but presume they showed evidence of hypertension.

Before the Workman's Compensation Court, I testified that in my opinion the fibrillation was due to a heart injury from the severe blow. I quoted Lewis who had made the statement in his book that fibrillation can result in cardiac hypertrophy. The man had been examined, upon being employed, a few months before and presumably did not have hypertension at that time. I reasoned that the blow had instituted the chain of events that resulted in his death but must admit that I was indulging in *post hoc propter hoc* reasoning. I was alone in my testimony that the blow had produced an injury to the heart, and the court's decision in favor of the plaintiff's widow may have been simply granting her the benefit of a doubt.

About this same time, Dr. E. L. Touhy, of Duluth, published in MINNESOTA MEDICINE reports of two cases of myocardial injury due to blows.

DR. JOHN F. NOBLE, Saint Paul: I came here this evening to listen to this thesis with a good deal of trepidation for I have listened to so much irrational, if not dishonest, medical-legal testimony on the subject that I was afraid I might be in total disagreement with the essayist. I find that I am in complete accord with Dr. Lowry's opinions on the subject. There is no question, as illustrated by the cases reported by Dr. Lowry, that trauma can cause acute and often fatal damage to the heart; but I feel very strongly that trauma is seldom, if ever, a cause of chronic heart disease.

DR. THOMAS A. PEPPARD, Minneapolis: I was impressed by one of Dr. Lowry's conclusions, i.e., the one relating to the attitude of the medical attendant. The situation in these cases of cardiac injury is quite similar to that in coronary thrombosis. The physician may induce or aggravate the anxiety of the patient, and set the stage for the "post thrombotic syndrome" (iatrogenic heart disease). The patient is often so terrified that he is just as miserable from his neurosis as he was from his acute myocardial infarction.

DR. S. R. MAXEINER, Minneapolis: I would like to call attention to an act of the Minnesota Legislature passed a few years ago which very greatly liberalizes the attitude toward compensation. We have had experience with several patients who had abnormal hearts, who sustained an aggravation by lifting while on duty. In several instances, they have been awarded compensation on the basis of an aggravation of a previously existing condition. It became necessary to retire one employe with cardiac disease whose condition became aggravated while on duty. In this state, the aggravation feature becomes extremely comprehensive and often constitutes a death claim.

DR. LOWRY, in closing: I have nothing further to add. I would like to thank all the discussors for their contributions. I was interested in what Dr. Fahr had to say about litigation. In looking over the literature, I found that in several case reports the results of the litigation were included as part of the medical record.

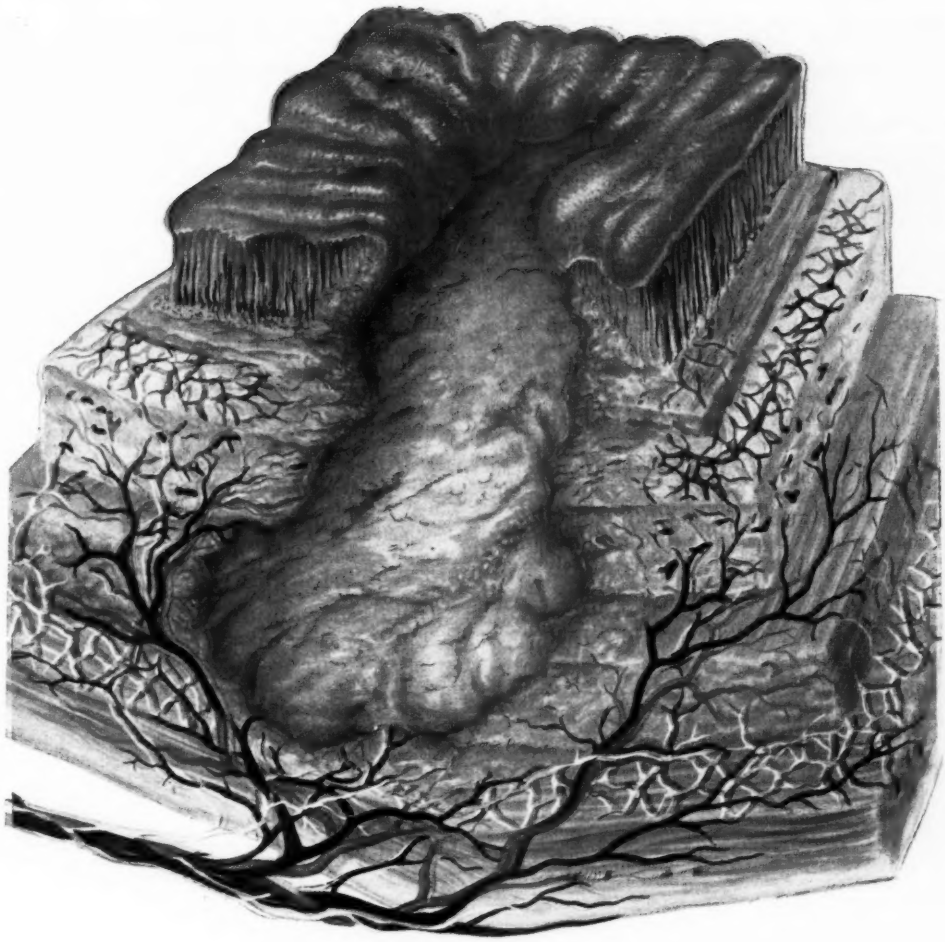
The meeting was adjourned.

WALLACE P. RITCHIE, M.D., *Secretary*

Despite all the problems inherent in the situation, past and present, there has been measurable progress in the control of tuberculosis among Indians. The most noticeable and important advance has been in the active participation of the Indians themselves through the medium of tribal health councils and tribal councils in developing community consciousness of the cause and control of tuberculosis. Many of the tribes have passed ordinances governing tuberculous individuals and their movements in the environment of the reservation including appropriate penalties for nonobservance. Information has been spread to the Indians largely through the tribal councils and, were it not for the participation of these governing bodies, no effective disease control programs could develop.—H. DELIEN, M.D., and ARTHUR W. DAHLSTROM, M.D., *American Journal of Public Health*, May, 1951.

* * *

Few diseases have shown the continuous, yearly decline in mortality exhibited by tuberculosis. Mortality from tuberculosis continued to decline in 1949, when 39,000 deaths from all forms of the disease occurred in continental United States. The death rate is estimated to be 26.2 per 100,000 population, excluding the armed forces overseas.—EVELYN H. HALPIN and OTIS D. TURNER, *Public Health Reports*, May 4, 1951.



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SEARLE RESEARCH IN THE SERVICE OF MEDICINE

Reports and Announcements

AMA CLINICAL SESSION

The 1951 Clinical Session of the American Medical Association will be held in Los Angeles December 4 to 7, inclusive. It is being designed primarily for the general practioner, and therapy will be stressed. Subjects of interest to the specialist, however, will not be forgotten.

The four-day scientific program will include discussions and presentations on urology; general practice; general surgery; cardiovascular diseases; industrial medicine and surgery; eye, ear, nose and throat diseases; diseases of the chest, and neuropsychiatry.

Color television will again be utilized to demonstrate surgery, clinical treatment and examination procedure. Scientific exhibits will cover a wide field.

The scientific and technical exhibits will be located in the Shrine Convention Hall, adjacent to the Al Malaikah Temple where the lectures will be given.

Physicians are urged to make hotel reservations in advance by writing to Chairman, AMA Subcommittee on Hotels, 1151 South Broadway, Los Angeles 15, Calif.

COMMONWEALTH AND EMPIRE HEALTH AND TUBERCULOSIS CONFERENCE

The National Association for the Prevention of Tuberculosis, England, has announced that the third Commonwealth and Empire Health and Tuberculosis Conference will be held in London, July 8 through 13, 1952. Lectures and discussions will be given at Central Hall, London, on July 8 through 11, with visits to sanatoriums, hospitals and clinics on July 11 through 13.

Further information can be obtained from the Secretary-General, National Association for the Prevention of Tuberculosis, Tavistock House North, Tavistock Square, London, W.C.I, England.

OMAHA MID-WEST CLINICAL SOCIETY

The nineteenth annual assembly of the Omaha Mid-west Clinical Society will be held at the Hotel Paxton, Omaha, Nebraska, from October 29 through November 2.

The meeting will be addressed by national authorities on a wide variety of subjects of special interest to general practitioners as well as specialists. Clinics, round-table discussions and question-and-answer periods will also be utilized in giving variety to the meeting.

The assembly was the first to receive Class A rating by the American Academy of General Practice for post-graduate study credit.

Further information may be obtained by writing to the Executive Office of the society, 1031 Medical Arts Building, Omaha 2, Nebraska.

VAN METER PRIZE AWARD

The American Goiter Association again offers the Van Meter Prize Award of $300 and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The award will be made at the annual meeting of the association which will be held in Saint Louis, Missouri, May 1, 2 and 3, 1952, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten double-spaced copy in duplicate sent to the corresponding secretary, Dr. George C. Shivers, 100 East Saint Vrain Street, Colorado Springs, Colorado, not later than March 1, 1952. The committee, who will review the manuscripts, is composed of men well qualified to judge the merits of the competing essays.

A place will be reserved on the program of the annual meeting for the presentation of the prize award essay by the author, if it is possible for him to attend. The essay will be published in the annual *Proceedings* of the association.

CONTINUATION COURSES

Chest Diseases.—The University of Minnesota, together with the Minnesota Public Health Association, will present a continuation course in chest diseases to be directed to the lay people particularly interested in this field. The course will be held at the Center for Continuation Study on the campus of the University of Minnesota from October 22 to 24. The course will emphasize public health aspects of tuberculosis and other chest diseases.

Child Psychiatry.—The University of Minnesota will present a continuation course in child psychiatry for general physicians and pediatricians November 26 to December 1. Dr. Reginald S. Lourie, director of the Department of Psychiatry, Children's Hospital, Washington, D. C., and Dr. J. Franklin Robinson, director of the Children's Service Center of Wyoming Valley, Wilkes-Barre, Pennsylvania, will be the visiting faculty members for the course. Dr. Reynold A. Jensen, associate professor, Departments of Psychiatry and Pediatrics, University of Minnesota, is chairman for the course.

MINNESOTA HEART ASSOCIATION

First grant of funds by the Minnesota Heart Association for research has proved effective.

Finances for detailed study of heart conditions were given a year ago to the University of Minnesota medical department for use in 1950-51.

Gratifying results "beyond original expectations" have been reported by the executive committee of the Heart Association. Their conclusions were based on reports made by each researcher at the end of his year's study.

From these reports the executive group made the statement that "the future appears still more promising from the viewpoint of scientific productivity."

"Part of the funds received from the Minnesota Heart

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Association have been used for equipping and staffing the laboratories designed for the investigation of various aspects of the rheumatic fever problem at the Heart hospital," one of the research reports pointed out.

Confirmation of the practical and effective use of last year's grant was given in a letter from Dr. Harold S. Diehl, dean of medical sciences at the University, to the Heart Association.

"The support received from the Minnesota Heart Association has been invaluable," he wrote.

A grant of $25,000 was made to the University for 1950-51. That sum was then distributed by an allocations committee of the medical department to members of the faculty to finance research in various fields of heart disease.

These grants by the Heart Association are financed from the subscriptions made in February each year to the Minnesota Heart Fund by individuals and organizations interested in the medical profession's daily fight against the nation's No. 1 killer, heart disease.

"This report by the executive committee is intended to inform the public how their money is being spent in the fields of research at the University of Minnesota," said Dr. Frank J. Hirschboeck of Duluth, president of the Heart Association.

The complete executive committee report follows:

"During the past year the Minnesota Heart Association has provided the major financial support for ten important cardiovascular research projects carried out by members of the University of Minnesota faculty. A

wide range of problems has been covered. Some of the studies have already produced definitive results justifying reports in the standard medical journals. Others have been of preliminary character, but have paved the way for more intensive investigations in the future.

"One of the latter group has dealt with the nature of the physiological adaptive mechanism which makes it possible for the child with severe cyanotic heart disease to withstand extremely high degrees of oxygen unsaturation of the arterial blood. Preliminary studies suggested the possibility that the hemoglobin of such patients may be of the fetal type, but conclusive data are yet to be obtained. The red blood cells were found to be more fragile than normal in the cyanotic child.

"Lesions in the heart simulating the clinical type of mitral stenosis were produced experimentally in dogs. Atrial and pulmonary vein hypertension resulted. This hypertension was reduced by shunting the blood from one atrium to the other by surgical means.

"Studies of the bacteriology of endocarditis and endarteritis previously shown to follow surgical production of extensive arteriovenous anastomoses were continued.

"The rheumatic fever problem was approached from various viewpoints. One indicated that the C-reactive protein occurs regularly in the serum of patients with rheumatic fever in an active phase, even when the sedimentation rate and other signs are equivocal. Its absence tends to rule out rheumatic fever in favor of other disorders. Unlike the increase in hyaluronidase inhibitor level, C-reactive protein did not appear to represent a response to the alarm reaction, according to results of this study.

"In studies on the effects of cortisone and adrenocorticotropic hormone in animals treated in various ways, it was found that the intravenous injection of endotoxin from gram-negative bacteria in cortisone-treated rabbits results in bilateral cortical necrosis of the kidneys, while intradermal injection of toxin in treated rabbits causes local hemorrhage in the skin site. Cortical necrosis could not be produced in this manner in animals pretreated with nitrogen mustard. While cortisone was shown to favor the development of extensive infections with Group A hemolytic streptococci in normal rabbits, it had no enhancing effect upon such infection in animals which had been immunized, indicating that the hormone does not block the mechanism of immunity but interferes with the little known mechanism of natural immunity. Such immunized animals were found to be capable still of developing extensive myocardial inflammatory lesions during cortisone treatment.

"A serological study in families of rheumatic fever patients has been initiated to determine if there might not be alterations in certain blood constituents indicative of rheumatic fever susceptibility. For example, such factors as mucoprotein and hyaluronidase inhibitor in the blood and 17-ketosteroids in the urine are being investigated. Follow-up studies of rheumatic fever patients showed the hyaluronidase inhibitor values in the serum to be low.

"In a comparative study on the body fluid space changes in normal and hypertensive subjects, it was found that the fluid compartments were normal in the latter. The decrease in blood pressure induced by excessive sweating or salt-restriction regimens appears from these results not to be due solely to changes in body fluid content.

"An investigation of the effects on cardiac function of breathing atmospheres high in CO_2 content revealed that heart muscle can tolerate high degrees of acidity surprisingly well. No electrocardiographic abnormalities were produced when the alveolar CO_2 was increased to high levels or when again reduced to normal levels gradually over a period of thirty minutes. When it was reduced too suddenly, however, ventricular fibrillation ensued with lethal termination in the majority of animals tested.

"The metabolism of heart muscle was studied in one project by use of isotopic carbon (C_{13}) incorporated into molecules of various organic acids. It was found that the labeled carbon from lactic, acetic and proprionic acids appeared in the respiratory CO_2 but not in cardiac glycogen. Succinic acid was not metabolized by the isolated heart.

"Preliminary studies to evaluate the usefulness of the new technique of electrocardiovectography have been carried out under a variety of conditions. The groundwork of standardizing techniques for normal subjects and for patients with such disorders as pulmonary emphysema and mitral valvular disease has been carried out and new apparatus has been devised for use in future work.

"Altogether, results of the first year of research, made possible by financial aid from the Minnesota Heart Association, have been gratifying beyond original expectations. Judging from preliminary reports recently rendered by recipients of the association's grants, the future appears still more promising from the viewpoint of scientific productivity."

UNIVERSITY OF MINNESOTA

Presents

A Symposium on Rheumatic Fever
Sponsored by the Minnesota Heart Association
November 29-December 1, 1951

PROGRAM

Thursday, November 29

Morning Session:

Greetings—Dean Harold S. Diehl, University of Minnesota Medical School.

Introductory statement on purpose and scope—Dr. Irvine McQuarrie, University of Minnesota Medical School.

Chairman: Dr. Irvine McQuarrie

The Natural History of Rheumatic Fever—Dr. T. Duckett Jones, The Helen Hay Whitney Foundation, New York, N. Y.

The Epidemiology of Rheumatic Fever—Dr. F. S. Schwentker, The Johns Hopkins Medical School.

The Pathology of Rheumatic Fever—Dr. George Murphy, Rockefeller Institute, New York.

Non-Rheumatic Myocarditis—Dr. Horace Hodes, Mount Sinai Hospital, New York.

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Afternoon Session:
Chairman: Dr. Lewis Thomas

Infection and Re-infection with Hemolytic Streptococci in Early Childhood — Dr. Lowell T. Rantz, Stanford University Hospitals, San Francisco, California.

Epidemiological Studies in the Armed Forces—Dr. Charles Rammelkamp, Western Reserve University, Cleveland, Ohio.

Immune Reactions in Rheumatic Fever—Dr. Maclyn McCarty, Hospital of the Rockefeller Institute, New York, N. Y.

Rheumatic and Other Inflammatory Complications of Congenital Heart Disease—Dr. Thomas J. Dry, Mayo Clinic, Rochester, Minnesota.

Subacute Bacterial Endocarditis—Drs. Wallace E. Herrell and Fordyce R. Heilman, Mayo Clinic, Rochester, Minnesota.

Friday, November 30

Morning Session:
Chairman: Dr. Jerome T. Syverton

The Biology of the Hemolytic Streptococcus — Dr. Maclyn McCarty, Hospital of the Rockefeller Institute, New York, N. Y.

Tissue Factors in Streptococcal Infection—Drs. Dennis Watson, and William Cromartie, University of Minnesota Medical School.

Review of Attempts to Induce Rheumatic-like Disease in Experimental Animals — Dr. George Murphy, Rockefeller Institute for Medical Research, New York, N. Y.

Streptococcal Infection and Cortisone — Dr. Lewis Thomas, University of Minnesota Medical School.

Experimental Bacterial Endocarditis—Dr. C. Walton Lillehei, University of Minnesota Medical School.

Afternoon Session:
Chairman: Dr. Wesley Spink

"Acute phase" Serological Reactions in Rheumatic Fever—Dr. R. A. Good, University of Minnesota Medical School

The Biochemistry of Connective Tissue in Rheumatic Fever—Dr. Albert Dorfman, University of Chicago, Chicago, Illinois.

The Penetration of Antigens into Connective Tissue—Dr. Albert H. Coons, Harvard Medical School, Boston, Massachusetts.

Pathogenesis of Arthus and Shwartzman Reactions—Dr. C. A. Stetson, Streptococcal Disease Laboratory, Warren Air Force Base, Cheyenne, Wyoming.

The Shwartzman Reaction Involving the Heart—Dr. Lewis Thomas, University of Minnesota Medical School.

Evening:
Panel Discussion of Pathogenesis of Rheumatic Fever
Chairman: Dr. Alvin Coburn
Participants: Drs. McCarty, Dorfman, Jones, Kutt-

ner, Murphy, Thomas, Rammelkamp, and Syverton.

Saturday, December 1

Chairman: Dr. Thomas J. Dry

Surgical Treatment of Rheumatic Heart Disease—Dr. R. Varco, University of Minnesota Medical School.

Treatment of Rheumatic Fever with ACTH and Cortisone—Dr. A. Barnes, University of Minnesota Medical School.

Recent Developments in the Therapy of Rheumatic Fever—Dr. Ann Kuttner, Bellevue Hospital, New York City.

The Prevention of Rheumatic Fever—Dr. Charles Rammelkamp, Western Reserve University, Cleveland, Ohio.

General Management of Rheumatic Fever—Dr. Morse Shapiro, Los Angeles, California.

There will be no tuition fee for attendance at the Symposium.

The following will take part in the Symposium:

Dr. Arlie Barnes, University of Minnesota Medical School
Dr. William Cromartie, University of Minnesota Medical School
Dr. Albert H. Coons, Harvard Medical School, Boston, Massachusetts
Dr. Alvin Coburn, Northwestern University, Chicago, Ill.
Dean Harold S. Diehl, University of Minnesota Medical School
Dr. Albert Dorfman, Bobs Roberts Memorial Hospital for Children, Chicago, Ill.
Dr. Thomas J. Dry, Mayo Clinic, Rochester, Minnesota
Dr. Robert A. Good, University of Minnesota Medical School
Dr. Fordyce R. Heilman, Mayo Clinic, Rochester, Minnesota
Dr. Wallace E. Herrill, Mayo Clinic, Rochester, Minnesota
Dr. Horace Hodes, Mount Sinai Hospital, New York
Dr. T. Duckett Jones, The Helen Hay Whitney Foundation, New York City
Dr. Ann Kuttner, Bellevue Hospital, New York City
Dr. C. Walton Lillehei, University of Minnesota Medical School
Dr. Maclyn McCarty, Hospital of the Rockefeller Institute, New York
Dr. Irvine McQuarrie, University of Minnesota Medical School
Dr. George Murphy, Rockefeller Institute, New York
Dr. Charles Rammelkamp, Western Reserve University, Cleveland, Ohio
Dr. Lowell T. Rantz, Stanford University Hospitals, San Francisco, California
Dr. Francis Schwentker, The Johns Hopkins Hospital, New York
Dr. Morse Shapiro, Los Angeles, California
Dr. Wesley Spink, University of Minnesota Medical School
Dr. C. A. Stetson, Warren Air Force Base, Cheyenne, Wyoming
Dr. Jerome T. Syverton, University of Minnesota Medical School
Dr. Lewis Thomas, University of Minnesota Medical School
Dr. Richard Varco, University of Minnesota Medical School
Dr. Dennis Watson, University of Minnesota Medical School

Cook County Graduate School of Medicine

ANNOUNCES CONTINUOUS COURSES

SURGERY—Intensive Course in Surgical Technic, two weeks, starting October 22, November 5, November 26.
Surgical Technic, Surgical Anatomy and Clinical Surgery, four weeks, starting October 8, November 5.
Surgical Anatomy and Clinical Surgery, two weeks, starting October 22, November 19.
Surgery of Colon and Rectum, one week, starting October 15, November 26.
Esophageal Surgery, one week, starting October 15.
Thoracic Surgery, one week, starting October 8.
Gallbladder Surgery, ten hours, starting October 22.
Fractures and Traumatic Surgery, two weeks, starting October 8.

GYNECOLOGY—Intensive Course, two weeks, starting October 22.
Vaginal Approach to Pelvic Surgery, one week, starting November 5.

OBSTETRICS—Intensive Course, two weeks, starting November 5.

MEDICINE—Gastroenterology, two weeks, starting October 15.
Electrocardiography and Heart Disease, two weeks, starting October 22.

DERMATOLOGY—Intensive Course, two weeks, starting October 15.

UROLOGY—Intensive Course, two weeks, starting October 8.
Ten-day Practical Course in Cystoscopy every two weeks.

ROENTGENOLOGY—Diagnostic and Lecture Course, two weeks, starting November 5.

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NORTHERN MINNESOTA MEDICAL ASSOCIATION

At the annual meeting of the Northern Minnesota Medical Association at Virginia on August 24 and 25 the following officers were elected: Dr. G. A. Sather, Fosston, president; Dr. W. S. Neff, Virginia, vice president; Dr. C. L. Oppegaard, Crookston, secretary-treasurer.

Crookston was selected as the site for the 1952 meeting.

SOUTHERN MINNESOTA MEDICAL ASSOCIATION

At the annual meeting of the Southern Minnesota Medical Association at Rochester on September 10, Dr. W. A. Merritt, Rochester, was elected president of the organization. Also elected to office were Dr. H. J. Nilson, Mankato, first vice president; Dr. W. C. Bernstein, Saint Paul, second vice president, and Dr. C. F. Stroebel, Jr., Rochester, secretary-treasurer.

More than 125 physicians attended the one-day meeting.

When the conviction that tuberculosis is an exquisite infectious disease has become firmly established among physicians, the question of an adequate campaign against tuberculosis will certainly come under discussion and it will develop by itself.—From *The Aetiology of Tuberculosis* by Dr. Robert Koch, 1882. Translated by Berna Pinner and Max Pinner.

Woman's Auxiliary

CHAIRMAN ANNOUNCES "LEGISLATION CORNER"

Mrs. Herbert F. R. Plass

The state board of the Woman's Auxiliary to the Minnesota State Medical Association hopes members are pleased with the legislative card enclosed in the September *Newsletter*. Please keep it in a convenient spot and use it often. Show it, when possible, to friends and members of other club groups. Friends and acquaintances can help keep Congressmen aware that the Auxiliary is an alert and interested voting public.

The issue of socialized medicine is still alive. Perhaps members no longer hear about a compulsory health insurance, but if one watches, he will see that there is a continuous flow of bills being introduced to Congress to provide "free" medical care to more and more people. Throughout the year it will be the purpose of this "corner" to keep members informed of pending legislation, and what stand the American Medical Association has taken. From time to time, help will be needed in writing to Representatives and Senators. Auxiliary members should keep their eyes on this "corner," so that they will be advised promptly of bills that need their action.

WOMAN'S AUXILIARY HISTORY

Mrs. E. M. Hammes, Sr.

(Continued from September issue)

Members serving in the capacity of president following Mrs. H. B. Sweetser and Mrs. Harry P. Ritchie were:

Mrs. Fred Adair, Minneapolis	1925-26
Mrs. J. T. Christison, St. Paul	1926-28
Mrs. B. F. Davis, Duluth	1928-29
Mrs. John D. Lyon, Minneapolis	1929-30
Mrs. S. S. Hesselgrave, St. Paul	1930-31
Mrs. James Blake, Hopkins	1931-32
Mrs. Edward Schons, St. Paul	1932-33
Mrs. Adolph Passer, Olivia	1933-34
Mrs. M. Nordland, Minneapolis	1934-35
Mrs. F. J. Elias, Duluth	1935-36
Mrs. E. M. Hammes, St. Paul	1936-37
Mrs. J. F. Norman, Crookston	1937-38
Mrs. W. B. Roberts, Minneapolis	1938-39
Mrs. A. C. Baker, Fergus Falls	1939-40
Mrs. M. A. Nicholson, Duluth	1940-41
Mrs. J. J. Ryan, St. Paul	1941-42
Mrs. R. J. Josewski, Stillwater	1942-43
Mrs. F. S. McKinney, Minneapolis	1943-44
Mrs. Anthony J. Bianco, Duluth	1944-45
Mrs. E. V. Goltz, St. Paul	1945-46

Mrs. M. S. Henderson, Rochester.....1946-47
Mrs. J. A. Thabes, Brainerd.....1947-48
Mrs. H. F. Wahlquist, Minneapolis.....1948-49
Mrs. H. E. Bakkila, Duluth.....1949-50
Mrs. Charles W. Waas, St. Paul.....1950-51

The objectives of the State Medical Auxiliary are set forth in Article II of the Constitution:

"The object of the Auxiliary shall be: (1) Through its members, to extend the aims of the medical profession to other organizations which look to advancement in health and education; (2) To assist in the entertainment of national, state, district and county meetings; (3) To promote friendship among physicians' families; (4) To do such work as may from time to time be approved by the Minnesota State Medical Association."

During 1928 the American Medical Association met in Minneapolis, and the Minneapolis and St. Paul auxiliaries joined in entertaining the national Auxiliary.

In recent years the State Medical Association has urged members of the Auxiliary to inform themselves on current political bills which affect the medical profession, and to endeavor to pass this information on to the laity through the various women's clubs to which they belong.

The Minnesota State Medical Association gives us splendid support and assistance and has confidence in our ability to help them.

Since 1931 we have been co-sponsors with the Minnesota Public Health Association for the tuberculosis radio contest. The best essays written by high school students are broadcast, and the award is given by the president of the State Medical Auxiliary.

The radio has also been used by several county auxiliaries as a means of educating their own members and the public on health subjects.

During the past five years, weekly radio programs have been broadcast from Worthington, using the American Medical Association platters.

Minnesota has worked hard to promote the sale of *Today's Health* which now has in our state 206 one-year subscriptions, 202 two-year subscriptions, and nineteen three-year subscriptions.

Subscriptions to the *Bulletin* have increased in the past year to 91.

Newsletters are published four times a year by the Minnesota State Medical Association and written by the Auxiliary for the purpose of keeping every doctor's wife informed as to what is being done, both in the state, and individually in each county auxiliary.

Our president and president-elect both attend the Presidents' Conference of the American Medical Association, and the president attends the annual convention of the American Medical Association.

We have a simple and practical legislative program which helps us to a better understanding of our state Constitution.

As to our philanthropies, they are many and varied, and the majority of them assist medical charities.

Christmas seals are sold by many county auxiliaries for the benefit of tuberculosis. Some auxiliaries assist in the heart drive, and we work for the Cancer Home and donate money to this worthy cause. We help with the mobile unit and work in the blood center. One auxiliary sponsors the Glen Lake Tuberculosis Sanitorium's sale of articles, which sale realizes a substantial sum of money for the patients.

Many auxiliaries collect medical and surgical supplies and send them to countries in dire need of these articles.

Many auxiliaries give public relations teas, at which time a good opportunity is afforded to have a health talk or show movies of interest to our non-medical visitors.

Scrapbooks for children's hospitals have been made by some auxiliaries, others have sent boxes for the needy to European countries.

(To be concluded in the November issue)

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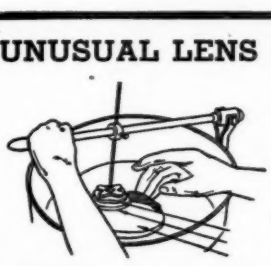

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In Memoriam

BENJAMIN FRANKLIN DAVIS

Dr. Benjamin F. Davis, formerly a Duluth surgeon, died August 26, 1951, at San Marino, California.

Dr. Davis was born in Chicago, October 13, 1884. He attended the University of Wisconsin and the University of Chicago where he received a Ph.D. in 1907. His M.D. degree was obtained at Rush in 1912, and he interned a year at the Presbyterian Hospital in Chicago.

After practicing seven years in Chicago, he moved to Duluth in 1920 where he engaged in private medical practice until 1942. In 1941 he was president of the St. Louis County Medical Society, of which he had been a member for years. He was also a member of the Minnesota State Medical Association and the American Medical Association, a member of the American Board of Surgery, a fellow of the American College of Surgeons and a member of the Western Surgical Association.

Dr. Davis is survived by his wife, Maria; a daughter, Sally, and three sons, William, John and Benjamin, Jr., all of San Marino, California.

HARRY B. EWENS

Dr. Harry B. Ewens, a prominent physician of Virginia, died on August 27, 1951, following a three months' illness. Dr. Ewens came to Virginia in 1910 to assist the late Dr. C. V. Malmgren, contract physician for the Oliver Iron Mining Company. Upon Dr. Malmgren's death, Dr. Ewen succeeded to the appointment. He was also a director of the Virginia school district for twenty-one years prior to his resignation in April, 1949, when he moved to Eveleth.

Dr. Ewens was born October 3, 1887, at Owen Sound, Ontario, Canada. He attended Toronto University where he obtained his M.D. degree in 1909. He also studied at McGill School of Medicine and took additional work at the University of Minnesota. His internship was served at Northwestern Hospital in Minneapolis. He served for a time in the Canadian Army overseas in Europe during World War II.

Dr. Ewens was a member of the More-Ewens Clinic and on the staff of the More Hospital at Eveleth. He was a member of the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association. Active in Masonic affairs, he was a past imperial potentate of the Duluth Shrine.

Dr. Ewens was married September 19, 1912, at Owen Sound, Ontario, to Marguerite Simpson who, with four sons, survives him.

THOMAS B. HARTZELL

Dr. Thomas B. Hartzell, prominent in the dental and medical circles of Minneapolis and the state, died August 26, 1951, at the age of eighty-five.

Dr. Hartzell was born in Beloit, Ohio, in 1866. He received his D.D.S. degree from the University of Minnesota in 1893 and his M.D. degree in 1894, also from Minnesota.

He had come to Minnesota in 1889 and taught in rural schools at Alexandria and Osakis, Minnesota. His teaching career began as an undergraduate instructor in histology at the University of Minnesota in 1890. He served as demonstrator in oral surgery from 1894 to 1896. From 1896 to 1903, he held the title of Professor of Comparative Dental Anatomy and Physical Diagnosis and Oral Surgery. From 1903 to 1913, he held a professorship in dentistry and in 1913 was made Research Professor in Mouth Infections. In 1920 he became a lecturer in Department of Medicine at the University.

Dr. Hartzell was president of the American Dental Association in 1921-22, a member of the General Medical Board of the Council of National Defense in 1917, a member of the medical section of the National Research Council and director of research in Minnesota for the American Dental Association.

Dr. Hartzell's training in both dentistry and medicine equipped him for the investigation of problems common to dentistry and medicine such as focal infections related to the teeth.

Dr. Hartzell was a member of the Delta Theta, Delta Sigma, Delta, Sigma Xi fraternities, the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association, and the American Dental Association. He was also a member of the Minneapolis Club, the Minikahda Club, the Lafayette Club, the Minneapolis Athletic Club and the Masons.

Dr. Hartzell is survived by his wife, Maud B.; a son, Dr. John B. Hartzell of Detroit, Michigan, and a daughter, Mrs. Walter Severson of San Francisco, California.

STANLEY G. LAW

On Monday, September 3, 1951, Dr. Stanley G. Law died of coronary heart disease at his summer cottage near Siren, Wisconsin. Dr. Law was a graduate of the University of Wisconsin, Rush Medical College, class of 1930. He was in general practice in Napierville, Illinois, until 1942 when he entered military service. He served as neuropsychiatrist with the U. S. Engineers on the Alaska-Canadian highway and as psychiatrist with the Manhattan Project in Oak Ridge, Tennessee. He left service with the rank of Major, Medical Corps. During 1945-1946, he was industrial psychiatrist to the Tennessee Eastman Corporation at Oak Ridge. In July, 1946, Dr. Law, with Drs. Eric Kent Clarke and William Fleeson, founded the Minnesota Psychiatric Institute in Minneapolis where he remained until the time of his death. He is the author of "Therapy Through Interview" published in 1948.

Dr. Law was a member of the American Psychiatric Association, The Association for the Advancement of Psychiatry, Hennepin County Medical Society and the Minnesota State Medical Association.

Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance. [1,2] Such states may involve any one of the organ systems or several at one time. [1,3] The outline below is designed to relate gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	Parasympathetic
Gastro-intestinal System	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio-vascular System	Rapid heart rate Peripheral vaso-constriction	Slow heart rate Vasodilatation
Functional Manifestations	Palpitation Tachycardia Elevated blood pressure Dry mouth and throat	Heartburn Nausea-vomiting Low blood pressure Colonic spasm

The data here tabulated is from references [3,4,5,6,7], given below.

When the clinical picture is suggestive of functional disorder, the diagnosis is supported by the presence of the following indications of autonomic lability:

Variable Blood Pressure
Body Temperature Variations
Changing pulse rate
Deviations in B. M. R.
Exaggerated Cold Pressure Reflex
Oculo-Cardiac Reflex Abnormalities
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relieving the somatic disturbance to prepare the patient for psychotherapy*; 2) guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

*Drug treatment using adrenergic and cholinergic blocking agents in conjunction with sedatives. [8,9,10].

1. Ebaugh, F.: Postgrad. Med. *4:* 208, 1948. 2. Wilbur, D.: J.A.M.A. *141:* 1199, 1949. 3. Williams, E. and Carmichael, C.: J. Nat'l. Med. Assoc. *42:* 32, 1950. 4. Goodman, L. and Gilman, A.: The Pharmacological Basis of Therapeutics, The Macmillan Co., 1941. 5. Katz, L. et al: Ann. Int. Med. *27:* 261, 1947. 6. Weiss, E. et al: Am. J. Psychiat. *107:* 264, 1950. 7. Alvarez, W.: Chicago Med. Soc. Bulletin, 581, 1950. 8. Rakoff, A.: A Course in Practical Therapeutics, Williams and Wilkins, 1948. 9. Karnosh, L. and Zucker, E.: A Handbook of Psychiatry. C. V. Mosby Co., 1945. 10. Harris, L.: Canad. M.A.J. *58:* 251, 1948.

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He is survived by his wife, Alda, two daughters, Jodene and Myrna, and a son, Walter. Dr. Law was buried at the Fort Snelling National Cemetery, Minneapolis, Minnesota.

HAROLD EDWARD RICHARDSON

Harold Edward Richardson died in Saint Paul on June 18, 1951, after a long and painful illness. He was born in Stevens Point, Wisconsin, on February 17, 1895. After residing in Saint Paul for a short time, the family moved to Minneapolis, where Dr. Richardson attended the public schools and graduated from East High School. He then entered the University of Minnesota and was graduated from the Medical School in 1919. In college he became a member of the Zeta Psi academic fraternity and the Nu Sigma Nu medical fraternity. He was dilligent, an excellent student, and was honored by being elected to Alpha Omega Alpha honorary fraternity.

Following his graduation from medical school, Dr. Richardson served as intern at the Minneapolis General Hospital, where he met Dr. Clarrissa Clay, who also was an intern at the hospital. Dr. Richardson and Dr. Clay were married on June 26, 1919. Their first child died in infancy, but two children, Ann and Harold, were born later and they survive him.

In 1920, Dr. Richardson became associated in the practice of internal medicine with the late Charles Lyman Greene in Saint Paul. Cardiology was Dr. Richardson's sub-specialty and his interest in this specialty thrived under the guidance of Dr. Greene.

In 1925, Mrs. Richardson began suffering from a series of illnesses which were agonizing not only to herself but to her husband. After her death in 1933, following a Cesarian section, Dr. Richardson's health broke. However, he refused to stop work. His resolute will and unfaltering devotion to duty, together with his deep-rooted faith carried him through many months of mental agony that would have overwhelmed a man of lesser caliber.

In 1926 Dr. Richardson became associated with the Saint Paul Clinic, where he remained for eleven years. In 1937 he began to practice independently and was in independent practice until the time of his death.

On April 17, 1937, happiness entered Dr. Richardson's life again when he married Margaret Horsch Kiesner, and two years later a son John was born. The family was a happy one.

In addition to his wife and children, Dr. Richardson is survived by three sisters, Marguerite Richardson and Mrs. Sophia Guernsey of Minneapolis, and Mrs. Grover Dimond of Saint Paul.

Dr. Richardson was a member of various medical societies including the American College of Physicians, the Central Society for Clinical Research, the Minnesota Society of Internal Medicine, the Ramsey County Medical Society, the Minnesota State Medical Association, the American Medical Association, and others.

The foregoing statistical data are woefully inadequate to convey a true picture of the personality of Dr. Richardson. "Hon," as he was known to his intimates, was a genial, devout, keenly intelligent, and tolerant man. He loved his fellow man, he loved music, he loved his work, and he loved life. We mourn his passing.

GORDON R. KAMMAN, M.D.

Of General Interest

The sympathy of the members of the Minnesota State Medical Association is extended to their executive secretary, **Mr. R. R. Rosell,** on the occasion of the loss of his wife on September 9. Mrs. Rosell had suffered a heart attack in June, at the time of the Minnesota State Medical Association meeting held in Rochester.

* * *

Dr. W. P. Freligh, Albert Lea, announced on September 7 that **Dr. Alfred G. Sherman** had joined the Surgical and Medical Clinic at Albert Lea on September 1. Dr. Sherman is specializing in the practice of ophthalmology. A graduate of the University of Minnesota Medical School in 1939, he was in general practice for a time at St. Peter. Before joining the clinic at Albert Lea he completed a three-year fellowship in ophthalmology at the University of Minnesota and Veterans Hospitals, Minneapolis.

* * *

Dr. Anne Kimball, director of the Division of Serology, Minnesota Department of Health, left in September for Rangoon, Burma, where she will teach serology in the Pasteur Institute as a member of a public health unit concerned with venereal disease and its relationship to maternal and child health. Dr. Kimball has been with the Minnesota Department of Health since 1932 and has been director of the Division of Serology since 1947.

* * *

In New Richland, **Dr. George H. Olds** is remodeling the lower floor of a newly purchased house into a modern medical office. He expects to move into the new office about November 1. When remodeling is complete, space will be available for an additional physician sometime in the future.

* * *

Dr. Philip K. Arzt, Saint Paul, has opened a new office at 1261 Lowry Medical Arts Building, Saint Paul. He is a diplomate of the American Board of Neurology and Psychiatry and limits his practice to this specialty.

* * *

The annual **Dr. Leo G. Rigler** Lecture in Radiology will be presented at the University of Minnesota on November 1 by Dr. W. Edward Chamberlain, professor of radiology at Temple University Medical School, Philadelphia. Dr. Chamberlain will speak on "Recent Advances in Equipment for Fluoroscopy" at 8:15 p.m. in the auditorium of the Museum of Natural History.

* * *

After five years of practice in association with **Dr. R. H. Puumala** in Cloquet, **Dr. W. B. Halme** moved to Wadena on September 4 and opened offices in the Imgrund Building. A graduate of the University of Minnesota Medical School in 1943, Dr. Halme interned at St. Mary's Hospital, Detroit, and then served an eighteen-month residency in obstetrics and gynecology at the Chicago Lying-In Hospital and Dispensary, University of Chicago.

* * *

The Minnesota Department of Health frequently receives information from various sources concerning pathologists and radiologists who are interested in relocating. Because of the shortage of specialists in these fields, communities have experienced difficulty in securing pathologists and radiologists. For this reason the Minnesota Department of Health is anxious to refer to interested groups such information concerning these specialists as may come to its attention. Inquiries should be directed to the Division of Hospital Services, Minnesota Department of Health, University Campus, Minneapolis 14, Minnesota.

* * *

The engagement of **Dr. John W. Donahoe** and Miss Joan Mary Bofenkamp was announced on August 30. Dr. Donahoe, a former fellow in the Mayo Foundation at Rochester, is now practicing medicine at Sioux Falls, South Dakota.

* * *

Dr. N. P. Blochowiak of Rushford has been appointed to serve on the Fillmore County Nurses Advisory Committee. The appointment was made by the county commissioners and is for a term of two years.

* * *

Among the grants awarded by the **Microbiological Institute** of the National Institute of Health is one for $12,000 to Theodore A. Olson, University of Minnesota, for a study of the toxic substances which may be produced in water supplies by certain plankton blooms, with special reference to those produced by blue-green algae.

* * *

Dr. and Mrs. Robert E. Fricke, Rochester, returned home on August 12 following a two-month tour of Europe. During the trip Dr. Fricke, who is with the section on therapeutic radiology of the Mayo Clinic, visited radium clinics in several foreign cities.

* * *

Dr. A. B. C. Knudson, former Veterans Administration medical officer in Minneapolis and now chief of the physical medicine and rehabilitation division of the VA in Washington, D. C., has been elected vice president of the American Congress of Physical Medicine.

* * *

Appointment of **Dr. Moses Barron,** Minneapolis, to the National Diabetes Detection Committee of the American Diabetes Association was announced on September 13. Dr. Barron will help formulate plans

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for Diabetes Week, November 12 through 18, when throughout the country testing of five million persons for diabetes will begin.

* * *

Dr. E. Q. Ertel of Ellendale was named "Man of the Week" by the *Albert Lea Tribune* in a feature story on September 2. Dr. Ertel, who moved to Ellendale in 1908 with the intention of staying only a year or two, has now been in practice there for forty-three years. Of him the Albert Lea newspaper said, "If there is such a thing as a pillar of a community, Dr. Ertel is that."

* * *

Dr. Robert Erickson, formerly of Portland, Oregon, and originally from St. Peter, is now associated in practice with Dr. J. J. Ahlfs at Caledonia.

* * *

Grants of more than $120,000 for the study of heart disease were awarded on September 8 to the **University of Minnesota** by the United States Public Health Service. The separate grants for heart disease studies and the men who will do the work are as follows:

Dr. Ivan D. Baronofsky, $16,541; Dr. E. T. Bell, $3,296; Dr. Ancel Keys, $30,000; Dr. Joseph T. King and Dr. Maurice B. Visscher, $17,998; Dr. John LaBree and Dr. Joseph Jorgens, $6,993; Dr. Walton C. Lillehei, $19,619; Dr. Otto H. Schmitt, $9,973; and Dr. Jerome T. Syverton, Dr. Dennis W. Watson and Dr. William J. Cromartie, $18,727.

* * *

In St. Cloud, **Dr. C. J. Luckemeyer** has moved his offices to 708 St. Germain Street.

* * *

Late in August, **Dr. E. J. Nelson,** after almost thirty years of medical practice in Owatonna, announced his temporary retirement because of ill health. He transferred his practice to **Dr. Daniel K. Halvorsen,** formerly of Minneapolis, whom he plans to rejoin in practice when his health permits.

Dr. Nelson, a graduate of the College of Medical Evangelists in Los Angeles in 1922, interned at Swedish Hospital, Minneapolis, before beginning practice at Owatonna. During his many years there he has been active in civic and professional affairs. He has served as president of the Steele County Medical Society and as chief-of-staff of the Owatonna City Hospital.

Dr. Halvorsen, who has been associated in practice with Dr. Nelson for almost a year, received his medical degree at Yale University and then interned at the University of Minnesota Hospitals. Previous to moving to Owatonna he was a fellow in surgery at the University Hospitals and a resident surgeon at Northwestern Hospital, Minneapolis.

* * *

Dr. Norval Westby, son of the late Dr. Nels Westby, has returned to Madison and has become associated in practice with the clinic there. A graduate of the University of Nebraska Medical School, he served his internship at Seattle, Washington, and then took a year of postgraduate work at the University of Wisconsin.

* * *

An open house was held at the Village hall in Waverly on August 26 in honor of **Dr. C. L. Roholt,** who was leaving Waverly. After thirty years of practice in the village, he was moving to McIntosh. The open house, which was attended by residents of the area to bid Dr. Roholt farewell and to thank him for his service, was sponsored by the Waverly Chamber of Commerce.

* * *

Dr. Philip S. Hench, head of the rheumatic disease department of the Mayo Clinic, Rochester, has been named one of twenty winners of the 1951 "Pennsylvania Ambassador" awards. The awards are given to native Pennsylvanians now living elsewhere who have made outstanding achievements. Dr. Hench, a Nobel Prize winner, was born in Pittsburgh.

* * *

It was announced in August that the Bemidji Clinic had acquired a new staff member. **Dr. John Wheeler Griffin, Jr.,** joined the clinic after moving to Bemidji from Manhattan, Kansas, where he had been in private practice.

Following his internship at Kings County Hospital, New York, Dr. Griffin served as a physician during the war in both the European and Pacific Theaters. After the war he completed a three-year residency at

King's Bridge Hospital, New York, and then served for one year at the Winter Veterans Administration Hospital, Topeka, Kansas. He is certified by the American Board of Surgery.

* * *

Dr. Max Seham, chief of pediatrics at Mount Sinai Hospital, Minneapolis, was one of the speakers at a meeting of the Women's League of Adath Jeshurun Synagogue in Minneapolis on September 4. Dr. Seham was a participant in a panel discussion on "Bringing Up a Normal Child in an Abnormal World."

* * *

Dr. Robert F. Conway, a former resident of Mankato, has become associated in practice with **Dr. E. G. Olmanson** in the St. Peter Clinic, St. Peter. A graduate of the University of Minnesota Medical School in 1948, Dr. Conway has since served in the Army. He interned at Letterman General Hospital, San Francisco, held a residency in surgery at Gorgas Hospital, Panama, until 1950, and then spent nine months at a station hospital at Spokane, Washington. For the five months preceding his move to St. Peter he was stationed at Topeka, Kansas.

* * *

Some statistics are now available on a **diabetic detection campaign** sponsored in Hibbing by the local Chamber of Commerce last winter. Sixty-seven per cent of the Hibbing population was screened in the survey which uncovered seventeen new diabetics and seven old cases. There were 11,062 tests conducted. The results, according to the American Diabetes Association, were almost identical to national percentages.

* * *

Dr. William B. Tucker, chief of tuberculosis at Minneapolis Veterans Hospital, attended meetings of the International Union Against Tuberculosis in Paris early in September.

* * *

Dr. Sol Austrian opened offices for the practice of medicine in Cosmos on September 6. A graduate of the University of Minnesota Medical School, he served his internship at the United States Marine Hospital at Galveston, Texas.

* * *

Dr. Walter A. Craychee, formerly of Minneapolis, has become associated in practice with **Dr. Roscoe C. Hunt** and **Dr. Robert S. Hunt** in Fairmont. A graduate of Temple University, Dr. Craychee interned in Detroit and then served in the armed forces for two years. He practiced medicine for two years at Mandan, North Dakota, after which he took postgraduate training at Northwestern Hospital and General Hospital in Minneapolis.

* * *

After twenty-three years of practice at Benson, **Dr. J. M. Arnson** has officially retired from the practice of medicine. He left Benson on August 20 for Eau Claire, Wisconsin, his place of birth, where he plans to make his permanent home.

Dr. Arnson, a graduate of Northwestern Univer-

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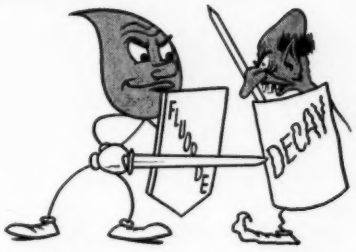

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sity Medical College in 1917, interned at Mercy Hospital in Chicago and at City and County Hospital (now known as Ancker Hospital) in Saint Paul. Following a year of service in the Army, he practiced at Graceville for seven years. He then spent a year in postgraduate study, both at schools in the United States and at the University of Vienna, Austria. He moved to Benson in 1928 and practiced there until the time of his retirement. Always active in civic and professional affairs during his years at Benson, he now plans to relax in his newly purchased home at Eau Claire.

* * *

Ground was broken on September 1 for a new nine-room office and clinic building which is being constructed by **Dr. D. C. Olson** in Gaylord. Modern in design, the one-story building will contain a waiting room, examining room, x-ray room, nurses' rooms, private offices. There will be enough space for another professional man in addition to Dr. Olson. The clinic is expected to be completed six months after construction was started.

* * *

Principal speaker at a county safety rally held at the Caledonia high school auditorium on September 25 was **Dr. H. Herman Young** of Rochester. Dr. Young showed color slides illustrating the results of farm accidents in southern Minnesota.

* * *

Dr. David J. Sanderson, formerly of Princeton, has joined the staff of the Fergus Falls Clinic. A graduate of the University of Minnesota Medical School, Dr. Sanderson served in the Navy for three years during World War II, then did postgraduate work in surgery at the University of Minnesota for three years. He practiced at Princeton for two years before moving to Fergus Falls on September 2. He is a diplomate of the American Board of Surgery.

* * *

Open house was held at a new medical building at Frost on September 9. The building was constructed by **Dr. Lewis Hanson,** who has been practicing at Frost since 1932.

* * *

Dr. Walter C. Alvarez, formerly with the Mayo Clinic, now living in Chicago, has been named editor-in-chief of *Modern Medicine,* a magazine published in Minneapolis. He succeeds **Dr. A. E. Hedback,** Minneapolis, the editor for twenty years, who has been named editor emeritus.

* * *

Dr. Ward Akester, a member of the staff of the Fergus Falls State Hospital for the past five years, has resigned to become a staff member of the Mount Pleasant State Hospital at Mount Pleasant, Iowa.

* * *

After twenty-one years of practice at Waterville, **Dr. C. A. Rohrer** has moved to Des Moines, Iowa, where he is specializing in radiology at the Veterans Hospital there. His move left Waterville with only one physician since he was unable to find anyone to replace him.

* * *

Principal speaker at a meeting of the Bethlehem Lutheran Mothers Club in Mankato on September 4 was **Dr. William S. Chalgren,** Mankato neuropsychiatrist. He discussed "Mental Health in the Child."

* * *

Minnesota's Professional Postgraduate Seminar has moved into its third year of successful operation. In the past two years the seminars, consisting of two lectures in each of eight evening sessions held once a week, have been presented at thirteen centers in Minnesota. The seminars for physicians cover the subjects of cancer, heart disease and psychosomatic medicine.

Five or possibly six centers are scheduled for seminars during the year 1951-1952. Red Wing and Brainerd are scheduled for this fall, Minneapolis and Saint Paul for this winter, and Faribault and perhaps one other city for next spring. The seminar staff is made up of University of Minnesota Medical School faculty members.

* * *

Dr. R. H. Puumala, Cloquet, coroner for Carlton County, discussed "Problems of the Coroner" at a meeting of the interns of St. Luke's Hospital, Duluth, on August 21.

* * *

Dr. George Earl, Saint Paul, was guest speaker at a meeting of the Kandiyohi-Swift-Meeker County

Medical Society in Litchfield on August 16. He spoke on "Problems in the American Medical Association" and showed motion pictures.

* * *

Dr. Robert A. Good, University of Minnesota Medical School, has been awarded a $6,825 research grant by the American Heart Association. Dr. Good specializes in the study of rheumatic fever.

* * *

The Medical and Chirurgical Society of Maryland will publish a monthly state medical journal beginning January, 1952. It will be known as the *Maryland Medical Journal* and will serve as the official organ of the state association. The new journal will be a member of the State Journal Advertising Bureau.

* * *

Dr. Benjamin A. Fine left Crosby during the middle of August and moved to California. Dr. Fine practiced in Crosby for ten years and was always active in numerous civic projects.

* * *

The foundation for the new **Mayo Memorial Medical Center** at the University of Minnesota has already been laid. Additional bids for the construction of the fourteen-story building were opened August 29, and the total accepted to date will bring the cost of the center to $11,609,990. Awards were made for the most part to Twin City contractors, and as soon as final approval of the United States Public Health Service is secured, the structural steel, already allocated by the Federal Security Agency, will start going up. This is expected not later than January 1, 1952.

The original intention was that the center would be twenty-two stories high. The State Legislature, however, which in 1945, 1947 and 1949 had appropriated $7,000,000 for the project, refused to appropriate the additional funds necessitated by increased costs to carry out the original plan. Two million dollars of the total were allocated by the United States Public Health Service from funds provided by the Hill-Burton Act, and the remainder came from grants from the National Cancer Institute, the National Heart Institute and private donations.

The new building will be a memorial to two outstanding Minnesota surgeons, Dr. William J. and Dr. Charles W. Mayo. In addition to the main building of fourteen stories, there will be an auditorium and a two-level garage.

* * *

Dr. Paul T. Lowry, who is studying internal medicine at the University of Minnesota, has been awarded a one-year postdoctoral fellowship by the National Foundation for Infantile Paralysis.

* * *

In St. Charles, **Dr. Pat Rollins,** formerly of Chicago, has moved into offices in a newly constructed office building. Dr. Rollins began practice at St. Charles during the past summer.

* * *

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Four Rochester physicians were among the guest speakers at the eighty-sixth annual session of the Michigan State Medical Society at Grand Rapids, Michigan, from September 23 to 28. **Dr. O. T. Claggett** spoke on mediastinal lesions; **Dr. James W. Kernohan** discussed peripheral neuritis; **D. H. L. Williams** presented a lecture on the medical treatment of Ménierè's disease; and **Dr. R. D. Pruitt** spoke on recent advances in the diagnosis of heart disease.

* * *

A limited number of **postdoctoral fellowships** to candidates whose interests are research and teaching in medicine and related biological and physical sciences are available, according to an announcement by the National Foundation for Infantile Paralysis. The fellowships cover a period of one to five years, with stipends ranging from $3,600 to $7,000 a year. Complete information concerning qualifications and applications may be obtained from Division of Professional Education, National Foundation for Infantile Paralysis, 120 Broadway, New York 5, New York.

* * *

Dr. D. M. Potek moved from International Falls on August 30 to join the medical staff of the Hastings State Hospital. A graduate of the University of Minnesota Medical School in 1933, Dr. Potek served his internship at St. Mary's Hospital, Duluth. After practicing briefly at East Grand Forks, he began practice at International Falls in 1936. In 1940 he joined the Army and served for five years before returning to his practice at International Falls.

* * *

The Charles Bolles Rogers achievement award, a silver bowl, to be known as the **St. Barnabas Bowl,** was presented to the Hennepin County Medical Society on August 28 by the board of trustees of St. Barnabas Hospital, Minneapolis. The award will be given annually to the outstanding doctor of the year in Hennepin County.

* * *

Dr. Hans Johnson, Kerkhoven, was honored for forty-six years of service in the area at a special Appreciation Day program in Kerkhoven on August 26.

A graduate of the University of Minnesota Medical School in 1904, Dr. Johnson practiced medicine at Murdock from 1905 to 1907, then moved to Kerkhoven, where he has since been in active practice. In his professional career he has served as health officer, as physician to the Swift County Selective Service Board, as president several times of the Tri-County Medical Society, and as chief-of-staff of both the Willmar and Benson hospitals.

Always active in civic affairs, he was the first president of the First National Bank of Kerkhoven, a charter member of the local Commercial Club, a charter member of the local Izaak Walton League, and is a member of several fraternal organizations. He has served on the village council and has twice been elected mayor of Kerkhoven. This year he began his thirty-ninth consecutive year on the school board, having served as its president continuously since 1927.

* * *

Dr. Chester W. Johnson, a resident of Excelsior, took charge of the Princeton Clinic at Princeton on August 30. It was announced that he would conduct the clinic for six weeks, at which time it was hoped that Dr. A. T. Kapsner would be able to resume his duties at the clinic.

* * *

The Public Health Service, through its chief research branch, the National Institute of Health, has awarded a total of 3,705 grants to non-Federal institutions and individuals, amounting to $44,371,188 during 1951. In the field of cancer, heart and mental disease $9,459,000 has been provided for special laboratory facilities, $8,100,000 to aid states in their disease control problems, $5,213,550 to improve and expand teaching facilities, and $1,693,315 to train physicians in new diagnostic or treatment procedures. Also $1,032,419 has been distributed to the states to initiate special control projects in cancer, heart disease and mental health. In the support of promising students doing postgraduate work in the field of medical research $1,568 321 was granted.

* * *

It was announced late in August that **Dr. Clinton G. Berg** and **Dr. Lewis M. Reid,** both of Excelsior, were planning to construct a new medical clinic this fall at Deephaven. Plans for the one-story clinic

called for facilities for three or four doctors, including rooms for examination, treatment, emergencies, x-ray, recuperation, laboratories, pediatrics and minor surgery.

* * *

Motion pictures, exhibits, educational programs and demonstrations, were among the activities that featured the **Freeborn County Health Week** from September 10 to 14. The event was the first countywide health program conducted in Minnesota.

Local and state physicians and other persons prominent in health activities participated in the week-long program. Although the major activities on the schedule took place at Albert Lea, motion pictures and talks were also presented at Alden, Freeborn, Emmons, Glenville and Hollandale. In addition, literature on numerous health topics was sent to each home in the county.

* * *

Dr. John S. Grogan, retired Wadena physician, was the subject of a feature story in the *Wadena Pioneer-Journal* on August 30. The story, which recounted incidents in the many years of service given by Dr. Grogan to the community, was one of a series on members of the Wadena board of education. Dr. Grogan has served on the board for eighteen years, taking a very active interest in all of its affairs.

HOSPITAL NEWS

Dedication of the new $357,000 **Buffalo Memorial Hospital** was held at Buffalo on August 26. Principal speaker at the dedication ceremonies was Dr. Ralph Rossen, Minnesota commissioner of mental health. The ceremonies wer followeed by an open house, with guided tours of the twenty-nine-bed hospital.

* * *

In Buffalo the **Catlin Hospital** closed on September 4 after thirty years of service in the community.

BLUE CROSS—BLUE SHIELD

Minnesota Blue Shield claims paid during August, 1951, amounted to $264,361.79. Analysis of claims paid in August, 1951, show that of a total of 7,850 claims, 7,567 involved services rendered within the current six month period—of these, 47 per cent covered services rendered in July, 1951, 18 per cent services in June, 18 per cent in August, 13 per cent covered services rendered from March through May, 1951, and the remaining 4 per cent were claims for services more than six months prior to the date of submission of the claim. Although this may appear as a relatively small proportion of claims over six months old, this percentage does have a direct influence on financial planning because there is no way of anticipating the number of old cases which may be presented in any given month. If this percentage continues over a long period of time, it represents a substantial portion of Blue Shield business.

The initiation of a Blue Shield claim is the subscriber's responsibility. Theoretically, the first portion of the report form is to be completed by the subscriber in the doctor's office where a supply of Blue Shield forms should always

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be on hand. In many cases, the doctor's office personnel has assumed the responsibility for completion of the subscriber's portion of the report as well as the description of service. However, the increasing number of telephone calls and written inquiries which are being received indicate that in many cases it is expected that Blue Shield will initiate the claim. Although the Blue Shield office endeavors to assist the doctor and the subscriber in every way possible, it should not be necessary in most cases to contact the Blue Shield office in the submission of a claim. If such inquiries and contacts can be kept at a minimum, more time can be devoted to the processing of claims.

The subscriber's identification card indicates whether or not Blue Cross-Blue Shield or both are in effect. If there are entries on the right hand side of the identification card under the effective date, the first figure designates the effective month of Blue Cross, the second figure indicates the day it went into effect, and the right hand box designates the Blue Shield coverage. If the right hand box is blank, the subscriber does not have Blue Shield coverage. On all Blue Cross claims, the identification card should be presented on admittance to the hospital.

During the month of July, Blue Cross paid out $910,457 to hospitals for Blue Cross subscribers' care and the total to date this year was $6,639,686. Administrative expenses have been reduced from 9.5 per cent in 1950 to 8.3 per cent in 1951. This has been accomplished through greater efficiency in the interest of economy, at the same time providing better service to subscribers, hospitals, and doctors. Blue Cross and Blue Shield employes total 320 compared with 420 a year ago.

All employes, equipment and records of the Minnesota Blue Cross-Blue Shield organization will move soon to the new, modern five-story Blue Cross-Blue Shield office building under completion at 2610 University Avenue in St. Paul. The increasing scope of Blue Cross-Blue Shield services finally outgrew the limited facilities of the present offices. Employes have been separated in two buildings, with many housed in a former storage building never intended for office occupation.

The new Blue Cross-Blue Shield headquarters will provide for the utmost efficiency on behalf of the public-at-large, the hospitals and the medical profession, as well as greatly improved working conditions for all employes. Arrangements are under way to give all doctors, their staffs and hospital personnel an opportunity to visit and inspect the new Blue Cross-Blue Shield building.

A LESSON FOR ALL

There is a lesson for all Americans, and not simply for the members of the medical profession, in the message which will be brought to Seattle next week by Leone Baxter and Clem Whitaker, general manager and director, respectively, of the American Medical Association's national educational campaign against socialization of medicine.

That lesson was epitomized in an address which Miss Baxter made some months ago at a conference at Tulane University, New Orleans.

In explaining how the physicians of America have successfully opposed administration efforts to regiment them, she observed:

"Our conception of practical politics is that if you have a sound enough case to convince the folks back home, you don't have to buttonhole the Senator. He will hear from home, and he is prone to respect very highly the opinions from that quarter."

The result was summed up by Mr. Whitaker, in a speech at the same New Orleans conference, in which he said:

"When doctors finally were able to convince other groups that they were not only fighting for self-interest, but that they were fighting for the health and welfare of the American people, their support mounted in spectacular fashion and new allies joined medicine's cause by the thousands."

While this team is coming to Seattle to address the Washington State Medical Association, it is to be hoped that other groups will have an opportunity to hear them. For theirs is the story of how a vital phase of the American way of life is being defended in a truly American fashion. — *Post-Intelligencer,* Seattle, Sept. 5, 1951.

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BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

CLINICAL-UNIPOLAR ELECTROCARDIOGRAPHY. Bernard S. Lipman, A.B., M.D., Assistant Professor in Medicine, Emory University School of Medicine, Atlanta, Ga.; and Edward Massie, A.B., M.D., F.A.C.P., Assistant Professor of Clinical Medicine, Washington University School of Medicine; Director of Heart Station, Barnes Hospital, St. Louis; Director, Cardiovascular Clinic, Washington University Clinics. 232 p. Illus. Price $5.00. Chicago: Year Book Publishers, 1951.

Recent advances in electrocardiography have brought about an increase in the number of books published concerning this subject. These volumes have varied from those that are elemental to those that are highly technical in character.

The present volume takes a pleasant middle of the way approach to the problem of teaching electrocardiography. The authors write clearly and simply; the illustrations as a whole, are well chosen and the format is good. Such criticism as may be directed is overweighed by the general quality of the book.

I can recommend it for those who are interested in a concise, well-documented volume.

JOHN F. BRIGGS, M.D.

OCCUPATIONAL THERAPY SOURCE BOOK. Edited by Sidney Licht, M.D. With an introduction by C. Charles Burlingame, M.D. 90 pages. Price $1.00. Baltimore: Williams & Wilkins Co., 1948.

Three years ago this little book was written and compiled for all who are interested in occupational therapy and mental hygiene and particularly for occupational therapists.

Dr. Licht, who is a specialist in physical medicine and rehabilitation, first outlines the early history of occupational therapy, in which he sets forth concisely and chronologically the various outstanding contributions that have been made through the years to humane care of the mentally ill from the time of Asclepiades, more than one hundred years before Christ, to the year 1917, when George Barton coined the phrase *occupational therapy.*

The editor then presents to the reader excerpts from a few of the most important writings on the subject that have appeared since the time when Pinel seized the opportunity to revolutionize the treatment of the insane during the French Revolution. All but one of the articles were published in the 19th Century. They include selections from the writings of Pinel, Reil, Hallaran, Rush, Tuke, Esquirol, Leuret, Voisin and Kirkbride. The 20th Century article selected is by Eva Charlotte Reid and, in the editor's estimation, "was one of the most important contributions in the field to appear before the first World War and ranks with the writings of Dunton." Each selection is prefaced with brief remarks about the author, when published, and why chosen to be included.

THE MANAGEMENT OF FRACTURES, DISLOCATION, AND SPRAINS. By John Albert Key, B.S., M.D., St. Louis, Missouri, Clinical Professor of Orthopedic Surgery, Washington University School of Medicine; Associate Surgeon, Barnes, Children's, and Jewish Hospitals; and H. Earle Conwell, M.D., F.A.C.S., Birmingham, Alabama, Associate Professor of Orthopaedic Surgery, University of Alabama School of Medicine; Chief of the Orthopaedic Service, South Highland Infirmary; Consulting Orthopaedic Surgeon to Carraway Methodist Hospital and Baptist Hospitals; Attending Orthopaedic Surgeon, Children's Hospital, Jefferson-Hillman Hospital, East End Memorial Hospital, and St. Vincent's Hospital, Birmingham, Alabama. 5th ed. 1232 p. Illus. Cost $16.00. St. Louis: C. V. Mosby Co., 1951.

The fifth edition of this widely known and highly popular reference book on orthopedic injuries has undergone considerable revision, but the general content and format closely resemble those of the fourth edition.

The sections on medicolegal problems and workman's compensation have been deleted from the book entirely. Perhaps the most striking change is in the section on low back injury. Dr. Key has removed the section dealing with low back strain for he believes that almost all such lesions are disc lesions. The revised discussion of disc lesions is excellent, both from the point of view of diagnosis and treatment. In the section on treatment the non-operative method of handling discs is presented

in meticulous detail so that no one reading the book should have any trouble using his method. The operative method is likewise handled well.

Other additions of note include information on the use of antibiotics and the use of intramedullary nails in fixation of fractures. These subjects are dealt with briefly and not in any detail but for a text of this type this treatment seems adequate. A discussion of the Putti-Platt and Baukart procedures in the treatment of recurrent dislocation of the shoulder is a welcome addition to the text. A similar improvement exists in the changes made in the section on anatomical consideration of hip injuries.

The deletions from the text have balanced the additions so that the fifth edition is about 100 pages shorter than the fourth edition. The text does not suffer from the loss of these sections, rather the book has been improved in its revision. A few minor errors exist in the index and in the text data, but in a work of this size this is difficult to prevent. It is a text to be recommended highly, especially to the general surgeon and general practitioner.

FREDERICH M. OWENS, JR., M.D.

HANDBOOK OF NUTRITION: A SYMPOSIUM. Prepared under the auspices of the Council on Foods and Nutrition of the American Medical Association. Second edition. 717 pages. Price $4.50. New York, Philadelphia, and Toronto: Blakiston Co., 1951.

This volume is a reprint of a series of articles which were written by authorities in their fields for the AMA Council on Foods and Nutrition and which were published intermittently in the *Journal of the American Medical Association* from 1948 to 1950. There have been some additions. The articles form a symposium of four parts on nutrition. Part I deals with individual nutrients; Part II sets forth the nutritional needs of human beings of various ages and under various conditions; Part III discusses nutritional deficiencies; and Part IV has to do with foods and their nutritional qualities. The book contains new information on these subjects, information brought to light by unusual war conditions. Each chapter contains many references to works cited. A detailed index adds to the volume's usefulness.

For persons in those professions interested in nutrition the Handbook should be a useful tool in summarizing for them the recent findings in the subject.

HYPERTENSION: A Symposium held at the University of Minnesota on September 18, 19 and 20, 1950. Edited by Elexious T. Bell, M.D. 573 pages. Illus. Price $7.50. Minneapolis: University of Minnesota Press, 1951.

This volume contains the most complete collection of essays by eminent authorities, both in the field of experimental research and in clinical medicine. The various theories of etiology—the anatomical, the chemical and the endocrine—are summarized. The most recent advances in therapy, covering all phases of the practice of medicine—dietary, pharmacological, surgical and psychological—are included. There are several sections devoted to questions and discussions, giving the essayist an opportunity to clarify or defend his theory.

This symposium is dedicated to three of Minnesota's outstanding workers in this field, who have gained recognition throughout the medical world, Dr. E. T. Bell, Dr. B. J. Clawson and Dr. G. E. Fahr. This book can be considered an authority of our present-day knowledge of this controversial subject.

—C. K. WILLIAMS, M.D.

CARDIOVASCULAR DISEASE: FUNDAMENTALS, DIFFERENTIAL DIAGNOSIS, PROGNOSIS AND TREATMENT. Louis H. Sigler, M.D., F.A.C.P., Attending Cardiologist and Chief of Cardiac Clinic, Coney Island Hospital; Consulting Cardiologist, Rockaway Beach Hospital; Consulting Cardiologist, Menorah Home and Hospital for the Aged. 551 pages. Illus. Price $10.00. New York: Grune and Stratton, 1949.

This comparably condensed textbook is clearly and concisely written, and the author has included the latest methods of diagnosis and treatment. Because of the rapid advances in this field, however, one will look in vain for the prophylactic treatment of rheumatic fever with penicillin, the use of cortisone and ACTH, or the surgical treatment of mitral stenosis. Electrocardiography is not discussed because of an earlier treatise of this subject by the author. The book is, nevertheless, worthwhile reading for medical students or physicians desiring an up-to-date discussion of cardiovascular disease.

FROM THE HILLS: AN AUTOBIOGRAPHY OF A PEDIATRICIAN. By John Zahorsky, M.D. 388 pp. Illus. Price $4.00. St. Louis: C. V. Mosby Co., 1949.

"From the Hills" is the story of a man whose life encompasses the development of modern pediatrics. To those who have ever come under the influence of John Zahorsky, it is a fascinating view into the life, the hopes, and the dreams of this leader in pediatrics. But even more, this book recreates the development of this country in the past seventy-five years; it is a bit of Americana with a nostalgic human touch. John Zahorsky's interests were many, his professional success unquestioned, yet withal he remained the warm, sincere and conscientious physician. His fascination with the poets and poetry reveal a character that sought expression of the beauty of life itself.

This is a book that should attract layman and physician, for John Zahorsky's story is the essence of humaness and hope.

CHARLES L. STEINBERG, M.D.

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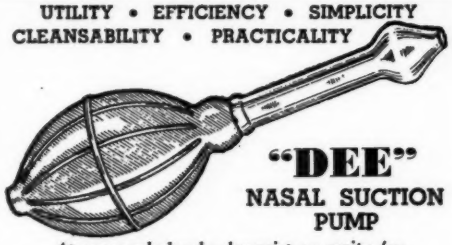

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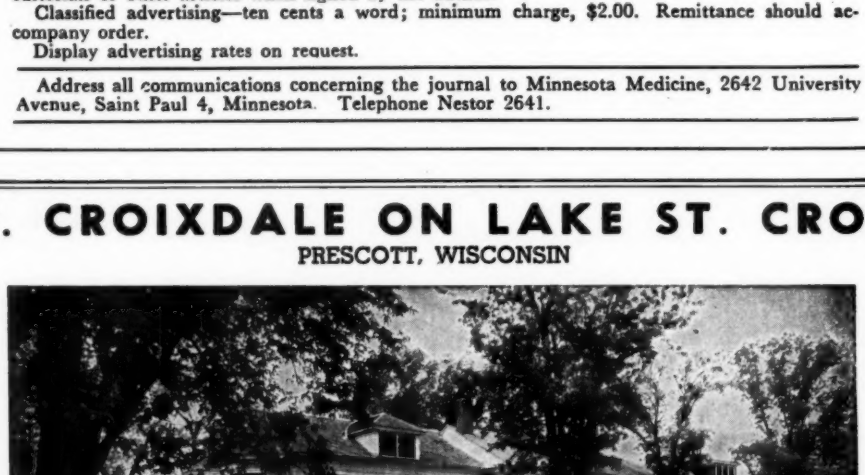

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